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MEDICAL NAMES IN AUSTRALIAN GEOGRAPHICAL NOMENCLATURE.[1]

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MEDICAL men play many parts in life. Some follow out their profession, others become explorers, soldiers, naturalists, novelists or men of letters. The monuments they leave are various—from a new name in surgery, such as Listerism, to delightful books, such as "The Autocrat of the Breakfast Table" and "The Vicar of Wakefield". But perhaps as enduring as all are those cases in which their names have been incorporated in geographical nomenclature, and will doubtless last while the places they represent continue their existence. In Australia, first and foremost, we have Bass Strait, so named after George Bass, Surgeon to the *Reliance*, who first discovered it. Dr. Leichhardt, the explorer, was a medical man and has left his name in a suburb of Sydney, whilst two other of its suburbs, Balmain and Redfern, derive their names from medical men. Charles Darwin and Napoleon's chief of the medical staff, Baron Larrey, also find places on the map of Australia. In the following lists other medical men will be found immortalized for reasons many and varied. In some instances they were persons of eminence or importance in the Australian communities of the earlier days; often they were personal friends of the explorers, or associated with them in some official capacity during some part of their lives. Sometimes they were contributors to the funds that enabled the discoveries to be made; in other instances they formed part of the personnel of the expedition. Or, again, they settled in or near the parts so named, and sometimes even towns finally

[1] Elaborated from a paper read before the Section of the History of Medicine of the South Australian Branch of the British Medical Association.

developed in the neighbourhood, which took their name from the adjacent squatter. Looked at from another point of view, whilst in the majority of instances the title to remembrance lay in the fact that they were medical men, in quite a number fame in some other capacity than that of medicine, for instance, as themselves explorers, or as naturalists, or as politicians, was the reason for their selection. We may, I think, indeed be proud of the services medical men have rendered in the development of Australia and deem them fully entitled to the modest additional fame and recognition entailed in placing their names on the map of Australia.

The appended list cannot be considered as at all complete, many names having doubtless been overlooked, to which it is asked that attention will be directed by those aware of the omissions. An analysis of those here presented shows that: In New South Wales 21 medical men have given their names to 25 places. In Victoria one medical man has given his name to one place, in Queensland 10 medical men have given their names to 14 places, in South Australia 8 medical men to 11 places, in Western Australia 28 medical men to 32 places, in the Northern Territory (including Central Australia) 15 medical men to 16 places. In Tasmania no places are called after medical men. Many of the names in the Northern Territory were given when this part was a portion of South Australia, and they might well have been included in that State's quota.

In connexion with the Federal Capital at Canberra, I am indebted to the Secretary of the Department of the Interior for the following note as to place names there.

> I desire to inform you that there are a number of place names in the Canberra City Area which are those of medical men. I am forwarding hereunder a list showing the origin of the names and the reasons why the names were adopted by the Canberra National Memorials Committee in the scheme of city nomenclature.
>
> Balmain Crescent, after William Balmain, M.D., R.N., pioneer surgeon on the first fleet (1788).
>
> Bass Gardens, after George Bass (1763-1808), navy surgeon, navigator, who discovered Bass Strait in 1798.
>
> Brown Street, after Robert Brown, F.L.S., F.R.S. (1773-1858), naturalist (army surgeon, 1795), noted for his botanical works concerning eastern Australia. (He accompanied Matthew Flinders as naturalist on the *Investigator* in 1800.)
>
> Mueller Street, after Baron Sir Ferdinand von Mueller, K.C.M.G., M.D., Ph.D., F.R.S. (1825-1896), famous for his botanical works on Australia and contributions to botanical science. Victorian Government Botanist, and naturalist on Gregory's expedition, 1856.
>
> Nicholson Crescent, after Sir Charles Nicholson, Bart., M.D., D.C.L., LL.D. (1808-1903), member of the first Legislative Council of Queensland, M.L.C. in New South Wales (1843), noted as a legislator and benefactor, and he took a prominent part in the foundation of Sydney University.
>
> Throsby Street, after Charles Throsby (1771-1828), navy surgeon, noted as the explorer of the Federal Capital Territory in 1820-1821.

Matthew Anderson, R.N., was appointed assistant surgeon at Sydney on February 18, 1824.

Anderson's Brook, New South Wales, the Severn of modern maps, was named by Allan Cunningham, the botanist and explorer, in July, 1827, "in compliment to my friend of the medical staff of the Colony".

Thomas Arndell (1752(?)-1821) was assistant surgeon to the first fleet. He arrived in New South Wales in January, 1788, and was the first medical man in Parramatta. He retired from office in 1792. He was appointed in 1802 a magistrate over the Hawkesbury, where he had a farm. Here, at Cattai Creek, he died in 1821, aged sixty-nine years [*vide* J. MacPherson, "Transactions of the Australasian Medical Congress (British Medical Association)", 1929, pages 195-198].

Arndell River, south of the Goulburn, was (says Hovell) named by Hume "in compliment to the late Dr. Arndell (the father of Mrs. Hovell) and to my own son, Arndell Hovell" (*Journal and Proceedings of the Royal Australian Historical Society*, Volume VII, Part 6, 1921, page 367).

Keith Ballow was for eight years, 1837-1845, Government Medical Officer for the Moreton Bay District.

Mount Ballow on McPherson Range, Queensland, and Ballow Street in the town of Coolangatta, Queensland, are named after him.

William Balmain, M.D., R.N. (1762-1803), was surgeon to the first fleet. He succeeded John White as Principal Surgeon to the Colony and its dependencies on May 17, 1797.

Balmain is a suburb of Sydney. The whole area of what is now the suburb of Balmain, 550 acres in extent, was granted to Balmain on April 16, 1800. On July 7, 1801, shortly before he was granted leave of absence to visit England, he alienated this, together with grants of similar amounts at Meadowlands and Windsor, to Dr. John Borthwick Gilchrist (who, it seems, was never in Australia), for, apparently, the sum of £17 10s., though the nominal sum of five shillings is mentioned in the conveyance. The reason for this transaction is not certainly known, though Sir Robert Ball suggests that it arose in discharge of a debt, due to Gilchrist in connexion with his banking operations, for the sum mentioned. Dr. Gilchrist was in India from 1785 till 1804, and died in 1841. On his return to England he founded a bank, which eventually formed, apparently, the nucleus of the Commercial Bank of Scotland. He bequeathed a sum of money to his trustees, the interest to be employed in any manner the trust deemed advisable for the diffusion of knowledge. As a result, the trustees founded the Gilchrist Lectures, and another part was devoted to scholarships, some of the latter money being expended in Australia. The land at Balmain formed part of this property. For long it was considered of no value, the trustees even hesitating as to whether they should incur the expense of fencing it. Eventually it was sold for £70,000. (S. Elliott Napier, *Journal and Proceedings of the Royal*

Australian Historical Society, Volume XIV, 1928, page 245.)

Thomas L. Bancroft, M.B., Ch.M., C.M.Z.S. (1860-1933), of Eidsvold, Queensland, was well known for his work in various branches of natural history and medical investigation and for the generous way in which he assisted other scientific workers with material.

Bancroft, a town in the Burnett district, Queensland, is named after him.

George Bass (1763-1808) was born in Lincolnshire. He was the son of a farmer and was apprenticed to a local surgeon. He obtained his diploma in London and then a commission in the Royal Navy as Surgeon to His Majesty's Ship *Reliance*, which sailed from Plymouth on February 15, 1795, with Governor Hunter and Matthew Flinders, who was a midshipman. He made boat expeditions with Flinders to Botany Bay and the Illawarra. In December, 1797, he left, this time without Flinders, on another boat journey and, reaching Wilson's Promontory on January 2, 1798, he was driven into Western Port by contrary winds. Surmising, from the rapidity of the tide and the swell, that there was a strait between Van Diemen's Land and the mainland, he and Flinders set out in a sloop, the *Norfolk*, in October, 1798, and circumnavigated what is now called Tasmania. On February 5, 1803, he sailed in a brig, *Venus*, for the coast of Peru. Nothing definite was heard of him from this date. It is believed the *Venus* was captured by the Spaniards and that Bass was sent a prisoner to the mines. There are reasons for thinking that he died in or before 1808.

Bass Strait was named by Governor Hunter at Flinders's recommendation on the return of the *Norfolk*. (Flinders, "A Voyage to Terra Australis", Volume I, 1814, Introduction, page cxciii.)

Point Bass, New South Wales, is at the south end of Shellharbour. "A long sloping projection which I have called Point Bass, lying about three leagues south of Alourie" (Flinders, *loco citato*, page cvi).

L. Becker was a member of the Burke and Wills exploration party in 1860 and was included in the party under Wright appointed by Burke. He died during the travels of this party in 1861, near Kooliatto Water Hole, on the Bulloo River.

The Parish of Becker, Queensland, is named after him.

Hugh Bell was surgeon of Flinders's ship the *Investigator*. Apparently he was not much of a social success amongst his fellow officers, as Flinders, in writing to Mrs. Flinders under the date June 25, 1803, at the conclusion of his explorations, said: "Mr. Bell is misanthropic and pleases nobody" (Scott's "Life of Matthew Flinders").

Point Bell, South Australia, in the Great Australian Bight, near Fowler's Bay, was named by Flinders in 1802 ("A Voyage to Terra Australis", Volume I, 1814, page 106).

Surgeon Major Bellefin was a member of the French expedition of 1801-1803.

Bellefin Prong, Western Australia, is named after him.

Alexander Berry (1781-1873) was a member of the Legislative Council of New South Wales. He passed through a course of study in preparation for the medical profession at Saint Andrews University and the University of Edinburgh. He entered the service of the Honourable East India Company as a surgeon and later engaged in mercantile enterprises. He first came to Port Jackson in 1808. In 1809 he visited New Zealand, reached Whangaroa and rescued a boy Davidson or Davison, Mrs. Mosely and her infant daughter, and a little girl, the daughter of Commissary Broughton, who afterwards married Charles Throsby, nephew of Dr. Charles Throsby. In 1822 he settled at Coolangatta, Shoalhaven, New South Wales. For sixty-six years he was a principal figure in the commercial and political life of New South Wales.

Berry, the town not far from Coolangatta, and Berry's Bay, Sydney, are named after him.

William Bland (1789-1868) in 1813 fought a duel with the purser of His Majesty's Sloop *Hesper* in which both were serving and, killing his adversary, was sentenced to transportation. Soon after reaching Sydney in 1814 he received a free pardon. He became a friend of Redfern and of W. C. Wentworth, was later returned to Parliament and became one of the most prominent practitioners in Sydney.

Bland's Plain, near Sunday Creek, south of the Goulburn, was named by Hume. (*Journal and Proceedings of the Royal Australian Historical Society*, Volume VII, Part 6, 1921, page 362.) Bland County and Parish in New South Wales, and Blandville, Hunter's Hill, Sydney, are named after him.

Sir Gilbert Blane, Bart., F.R.S. (1749-1834), was appointed physician to the fleet by Admiral Rodney during his West Indies expedition (1779-1783). "To his knowledge and attention it was owing that the English Fleet was, notwithstanding their excessive fatigue and constant service, in a condition always to attack and defeat the public enemy. In my own ship, the *Formidable*, out of 900 men, not one was buried in six months." In 1793 he recommended lemon juice, already known for a century, as a preventive of scurvy. In later years he rendered many services of importance to the Government, being frequently consulted on medical matters.

Point Blane, Northern Territory, was named by Flinders in 1803 after Sir Gilbert Blane, of the Naval Medical Board.

Charles Smith Bompas was born about 1818, retired 1886. He became M.R.C.S. (England) in 1840 and L.S.A. (London) in 1841 (*vide* F. I. Bray's account of "Some Early Medical Men of Western Australia").

Bompas Hill, Western Australia, was named after him.

Dr. Bowman was Government Medical Officer for the Moreton Bay District, 1831-1837.

The Parish of Bowman and Mount Bowman, Queensland, were named after him.

Robert Brown, F.R.S. (1773-1858), was born at Montrose, Scotland. He was educated at Marischal College, Aberdeen. He entered the Army in 1795 as ensign and assistant surgeon. He accompanied Flinders as naturalist in the *Investigator* from 1801 to 1803 in his celebrated explorations of the Australian coast. He then collected plants in New South Wales, Kent's Group and Tasmania, returning to England in 1805. He published portions of his *"Prodromus Florae Novae Hollandiae"* in 1810 and 1830. Mainly as the result of his Australian researches he became recognized as the most eminent botanist of his day.

Point Brown, South Australia, in the Great Australian Bight, near Streaky Bay, was named by Flinders in 1802. ("A Voyage to Terra Australis", 1814, page 110.)

Mount Brown, South Australia, a prominent mountain in the Flinders Ranges, near Port Augusta and Quorn, was named by Flinders in 1802.

Mount Brown, New South Wales, part of the Goulburn Range, between the Lachlan and the Macquarie, was named by Oxley on June 19, 1817.

Benjamin Bynoe, F.R.C.S., was surgeon to the *Beagle* (1837-1843); first under Captain John Clements Wickham, then under Captain John Lort Stokes.

Bynoe Ranges, Northern Territory, were named by Captain Stokes after Mr. Bynoe, who accompanied Lieutenant King in exploring the upper parts of the Victoria River in 1839. (Stokes's "Discoveries in Australia", Volume II, page 81.)

Bynoe Harbour, in Patterson Bay, near Port Darwin, is "quite an inner haven which was named after Dr. Bynoe", September 12, 1839. (Stokes's "Discoveries in Australia", Volume II, page 15.)

Bynoe River, Gulf of Carpentaria, Queensland, was named when he accompanied Captain Blackwood in the *Fly*, 1842-1846.

William Bell Carlyle (or Carlisle), R.N., (1788(?)-1844), surgeon superintendent of transport vessels, finally landed in Sydney in 1827 and settled on his grant of 1,000 acres at Invermain in the Hunter Valley. Later he joined the New England squatters. He moved to Port Macquarie in 1836, where he died. (*Journal and Proceedings of the Royal Australian Historical Society*, Volume VIII, 1922, page 260.) The tablet to him in Port Macquarie Church, where the spelling appears as Carlisle, runs as follows:

> Sacred to the memory of William Bell Carlisle Esqre.
> Surgeon in the Royal Navy and for many years
> a Magistrate of this Territory
> who departed this life on the 5th day of September, 1844,
> aged 56 years.
> This tablet is erected to his memory by his friends . . .
> *et cetera.*

This is evidently the Dr. Carlisle whom J. H. Maiden (*Agricultural Gazette of New South Wales*, Volume XXIII, 1912, page 713) mentions as having taken prickly pear to Scone as a rare plant in a pot "soon after 1839". If he was responsible for the introduction of this pest to that district it was doubtless before 1839, as he disposed of both his Hunter River and New England properties in 1836 before going to Port Macquarie.

Carlyle's Gully, a tributary valley of the Muluerindie River, New England, New South Wales, was occupied by him.

Dr. Chace or Chase. Dr. A. A. Lendon informs me that the name Chace or Chase, however spelt, does not appear in the Medical Register of South Australia, and that in Gwenneth Williams's Tinline Essay on "South Australian Exploration to 1856", page 85, it is stated that he "explored north of Arkaba (north-east of Hawker) in 1851".

Chace Range, north-east of Hawker, South Australia, was named in 1851.

William Lennox Cleland, M.B., Ch.M. (1847-1918), was Colonial Surgeon of South Australia.

Cleland Hills, north of Lake Amadeus, Central Australia, were named by W. H. Tietkins on May 14, 1889.

The Honourable Sir John Cockburn, K.C.M.G., M.D. (London, with gold medal, 1874) (1850-1929), came to South Australia in 1875 and settled at Jamestown. He became Premier (1889-1890) of South Australia at the age of thirty-eight, and later Agent-General.

Cockburn, the town in South Australia, was laid out in 1886 and called after Sir John Cockburn, at that time Minister for Education.

Dr. Alexander Collie, R.N., was born in Scotland. He arrived in Western Australia as surgeon of His Majesty's Ship *Sulphur* in 1829. He was appointed to act as Colonial Surgeon in 1832, and died at Albany in 1835 or 1836.

Collie River, Western Australia, was discovered by Dr. Collie and Lieutenant Preston, and named by Governor Sir James Stirling in 1830.

Charles Darwin, F.R.S. (1809-1882), was the famous naturalist.

Port Darwin (now Darwin), Northern Territory, was named by Captain Stokes on September 8, 1839.

> The point was called in consequence Talc Head. The other rocks near it were of a fine-grained sandstone, a new feature in the geology of this part of the continent, which afforded us an opportunity of convincing an old shipmate and friend that he still lived in our memory; and we accordingly named this sheet of water Port Darwin. (Stokes's "Discoveries in Australia", Volume II, page 6.)

Denmark was a British naval surgeon.

Denmark River, Western Australia, was so named by Dr. T. B. Wilson in compliment to a physician of the British Fleet. Dr. Wilson had received his first promotion in the service at the instance of Dr. Denmark. (Wilson's "Narrative of a Voyage Round the World", 1835, pages 261, 262.)

Henry Grattan Douglass, M.D., M.L.C. (1791-1865), served in the Peninsular War and in the West Indies. He arrived at Sydney from Britain in 1826. He was assistant surgeon at Parramatta and a fellow of the Senate of the University of Sydney. He was appointed to the Legislative Council in 1826. J. H. Maiden says that he was evidently a storm centre, and his removal from the magistracy by the Secretary of State was with Governor Brisbane's concurrence.

Douglas (*sic*) Park Railway Station (southern line, New South Wales) was named after him. He was an early settler in the district. (C. A. Irish, *Journal and Proceedings of the Royal Australian Historical Society*, Volume XIII, 1927, page 113.)

William Elgee, L.R.C.P. (London), 1890, M.R.C.S. (England), 1890 (1866-1906), arrived in Western Australia in 1892 and practised at Guildford and Midland Junction.

Elgee Cliffs, Western Australia, were named after him.

Charles Bolton Elliott, M.R.C.S. (England), L.R.C.P. (Edinburgh) (?-1904), was resident medical officer of the Geraldton district from 1874 to 1904.

Elliott Lake and Elliott Mountain, Western Australia. My information does not state whether these places were named after C. B. Elliott or after the following. They were not related.

Charles Henry Elliott, M.R.C.S. (England), 1858, L.S.A. (London), 1859, was resident medical officer, Colonial Hospital, Perth, 1874; he then held country appointments and finally was District Medical Officer, Champion Bay, 1886.

J. R. Elsey was surgeon and naturalist to A. C. Gregory's expedition.

Elsey Creek, a branch of the Roper River, Northern Territory, was named by Gregory in 1856 (?).

Sir Walter Farquhar, Bart. (1738-1819), studied at Aberdeen, Edinburgh and Glasgow. Without graduating in medicine, he entered the Army. He took part in Lord Howe's expedition to Belle Isle in 1761. He was afterwards stationed at Gibraltar. He took his M.D. degree at Aberdeen in 1796, was created a baronet in the same year, and shortly afterwards was appointed Physician-in-Ordinary to the Prince of Wales. (*Dict. Nat. Biogr.*)

Mount Farquhar, near the Lachlan, was named by Oxley on July 22, 1917.

John Ferguson, F.R.C.S. (Edinburgh), 1822 (1800-1881), was Colonial Surgeon of Western Australia from 1847 to 1872.

Ferguson River, Western Australia, was named by A. C. Gregory in 1852.

John Michael Gunson, M.R.C.S. (England), 1848, M.D. (Heidelberg), 1865 (1826-1884), was born in Limerick, Ireland. He arrived in South Australia in 1852, was registered by the South Australian Medical Board in April, 1853, and practised in Adelaide. He was one of the members of the first Council of the University of Adelaide and was admitted M.D., *ad eundem gradum*, in 1877, was appointed a member of the honorary staff of the Adelaide Hospital in 1867.

Mount Gunson, South Australia, seventy-five miles north-west of Port Augusta, was named by Surveyor H. Brooks about 1875.

Hamilton was surgeon of Captain Sturt's regiment (the 39th).

Hamilton Plains, beside the lower Murrumbidgee, were named "in remembrance of the surgeon of my regiment" by Charles Sturt in 1829.

John Stephen Hampton, F.R.C.S. (Edinburgh), 1828, was Governor of Western Australia from 1862 to 1868. During his *régime* many important public works were carried out, and his administration was one of the most successful.

Hampton Plains, Coolgardie district, Western Australia, were discovered by Surveyor C. C. Hunt.

John Harris (1754-1838) was surgeon of the 102nd Regiment. He was born in Ireland and qualified at Edinburgh. He served in the Navy in the East Indies. Appointed in 1789 surgeon-mate of the New South Wales Corps. Arrived in the *Surprise* in June, 1790. He became "naval officer" (controlling the port of Sydney), a magistrate and head of the police. He helped to found the Bank of New South Wales. In 1818 he volunteered to accompany Oxley as surgeon on the expedition in which he discovered the Liverpool Plains and Port Macquarie.

Harris Island, Northern Territory, near Melville Island, was named by Lieutenant Roe in October, 1824, "after my friend John Harris, Esqr., formerly surgeon of the 102nd Regiment, who has served so long and so faithfully in various offices under the Government of New South Wales" (King, "Discoveries", Volume II, page 237).

Mount Harris, west of New South Wales, was apparently called by Oxley after Dr. Harris, surgeon of the 102nd Regiment, who accompanied him in 1818. (Howitt, "The History of Discovery in Australia", Volume I, page 214.)

Joseph Harris (?-1847) was Colonial Surgeon of Western Australia from 1845 to 1847.

Harris River, Western Australia, was named by Surveyor A. Hillman in 1835.

Joseph Mountain, Western Australia, was named by Surveyor Hillmann in 1835, after Dr. Joseph Harris, at that time a member of his exploring party.

Dr. Hemsted, of Newbury. The Hemsteds are an old medical family in Hampshire. As Captain Roe's expeditions were carried out between 1830 and 1849, it is not clear which Dr. Hemsted, of Newbury, he honoured when he named Hempstead Hill, Western Australia (the spelling of the name as given by the Surveyor-General of Western Australia).

John Sydney Hicks, L.S.A., 1887, M.B. (London), 1888, M.D. (London), 1890 (1864-?), left England

for Western Australia in 1890. He represented Roebourne in the Legislative Assembly in 1900, and was Minister for Labour and Commerce, 1905-1906.

Doctor Hicks Range, Western Australia, was named after him.

H. Hines was a medical practitioner of Swan, Western Australia, about 1860.

Hines Hill, Western Australia, was named by Dempster and Clarkson.

Frederick Maurice House, M.R.C.S. (England), 1888, L.R.C.P. (London), 1888, lived at Beverley, Western Australia, and was later naturalist to F. S. Brockman, Kimberley exploration, in 1901.

House Mount, Western Australia, was named by F. H. Hann in 1898.

James Hunter, R.N., surgeon on Captain P. P. King's third voyage of exploration, commenced in 1820. In his first two voyages King had felt the lack of a medical man and had communicated with the Admiralty. As a result, a surgeon arrived with an appointment to the *Mermaid*, but to King's mortification he was found to be afflicted with mental derangement, which continued so long and so severely that he had to be sent back to England. At this juncture, a surgeon of the Navy, Mr. James Hunter, arrived in charge of a convict ship and volunteered his services, which were gladly accepted.

> The accession of a surgeon to our small party relieved me of a greater weight of anxiety than I can describe; and when it is considered that Mr. Hunter left an employment of a much more lucrative nature, to join an arduous service in a vessel whose only cabin was scarcely large enough to contain our mess-table, and which afforded neither comfort nor convenience of any description, I may be allowed here to acknowledge my thanks for the sacrifice he made. (King, Volume I, page 346.)

Hunter River opens into Prince Frederick Harbour in York Sound, north-west coast of Western Australia. On September 12, 1820, King found a spring of fresh water, supplying at the rate of two to three gallons a minute, amongst the mangroves where this stream entered the harbour—"a discovery so valuable that the river was thought worthy of a name, and it was called after my companion, Mr. Hunter, who shared my gratification at finding what we had thought, at this season, totally wanting near the coast" (King, Volume I, page 405).

Alexander Imlay arrived in the brig *Elizabeth* in 1829 at Hobart Town. He was appointed Assistant Surgeon, Medical Staff, Sydney, in 1830, and then engaged in extensive pastoral pursuits in the Goulburn and Twofold Bay districts. He died in Tasmania.

George Imlay, R.N., arrived about 1833. He was associated with Alexander and the third brother, Peter, in pastoral pursuits at Bega, and died near Bega in 1846. (For fuller accounts of the Imlay brothers, see Dr. A. A. Lendon, *Journal and Proceedings of the Royal Australian Historical Society*, Volume XVII, Part III, page 206, and H. P. Wellings, *loco citato*, Volume XVII, Part IV, page 209.)

Shire of Imlay, New South Wales, Imlay Street, Eden, New South Wales, Mount George, near Eden, were called after Dr. George Imlay. Imlay is a suburb of Wanganui, New Zealand.

The Honourable Adam Jameson, M.B., M.S. (Edinburgh), 1883, M.L.C., was Minister for Lands, Western Australia, in the Leake Ministry of 1901, and the succeeding James Ministry. He resigned in January, 1903, to accept the position of Commissioner of Crown Lands in the Transvaal. He was killed in a railway accident near Delagoa Bay, South Africa.

Jameson Mount, Western Australia, was named by F. S. Brockman in 1901.

Henry Truman Kelsall, M.S. (London), 1885, M.R.C.S., 1886, M.D. (London), 1888 (1865-1932), was a well known medical man and oculist of Perth, Western Australia.

Kelsall Lake, Western Australia, is named after him.

W. R. Kenny. Kenny Hill railway station, on the Campbelltown-Camden line, New South Wales, is named after him. He had been a resident in the district in 1834.

Archer Benjamin Kent, M.D. (Edinburgh), 1831, arrived in Adelaide in 1840, probably aged about thirty. He died at Ryde, in the Isle of Wight, in 1864. (A full account of what is known about him is given by Dr. A. A. Lendon in the *Proceedings of the Royal Geographical Society of Australasia*, South Australian Branch, Volume XXVII, Session 1925-1926, page 29; and Volume XXX, Session 1928-1929, page 174).

Sketch by Dr. A. C. Kelly, of Tintara Vineyard, McLaren Vale, showing Dr. Kent's house, now the suburb of Kent Town, Adelaide. (Reproduced by kind permission of Mrs. T. C. Angove, a grand-daughter of Dr. Kelly.)

Kent Town is a suburb of Adelaide, on which site he erected his house. The exact situation was later occupied by the Kent Town Brewery, and is in the direct continuation of Rundle Street, Adelaide, to the Parade, Norwood. There is a legend that Dr. Kent would not allow Rundle Street to be continued

on directly to the Parade, which accounts for the present deviation round the site of the brewery.

H. Landor was a medical practitioner. Landor River, Western Australia, was named by E. T. Hooley in 1886.

Baron Dominique Jean Larrey (1766-1842), the noted French surgeon, went first into the Navy and then into the Army. He accompanied Napoleon into Egypt in 1798, where he and Desgenettes established hospitals, and as Chief of the Medical Staff in the Moscow campaign of 1812 and 1813. He introduced the ambulance volantes, and published *"Mémoires de Médecine et Chirurgie,* 1812-1818," *et cetera.*

Larrey Point, Western Australia, was named by Baudin's French expedition of 1801-1803.

George Lawson, L.F.P.S. (Glasgow), 1835, J.P., arrived at Port Lincoln in 1844 and died as a result of falling off his horse in 1867.

Lawson Creek, near the Sturt Plains and Ashburton Range, Northern Territory, was named on May 14, 1861, by John McDouall Stuart.

Charles William Laver was born at Castlemaine, Victoria. He is L.R.C.P. and S. (Edinburgh), 1894, L.M. (Edinburgh and Glasgow), 1894, and was intimately associated with the first discoveries of gold in various parts of Western Australia, and is now resident at Kalgoorlie.

Laverton, a town near Mount Morgans, Western Australia, was named in 1900.

Frederick Wilhelm Ludwig Leichhardt (1813-1848), the famous explorer, migrated to New South Wales in 1841. He was lost sight of in 1848 in the western district of Queensland.

Leichhardt, a district 23° 50′ south, 148° 15′ east, in Queensland; Leichhardt, the well known suburb of Sydney; Leichhardt River (19° south, 139° 32′ east), running into the head of the Gulf of Carpentaria, named by A. C. Gregory in 1856; Leichhardt Range (20° 45′ south, 147° 30′ east), south-east of Bowen, Queensland, are named after him.

Richard Robert Madden (1798-1886), Colonial Secretary of Western Australia, took his M.R.C.S. diploma in 1849 and was elected fellow in 1855.

He was admitted to the intimate circle of Lady Blessington in 1823, and became a friend of Lord Byron and Count D'Orsay. He afterwards published "The Literary Life and Correspondence of the Countess of Blessington". He travelled much in the Levant; was one of the special magistrates appointed to administer the statute abolishing slavery in 1833 in Jamaica; became superintendent of liberated slaves; and was judge arbiter in the mixed court of commission at Havana. He accompanied Sir Moses Montefiore on a philanthropic mission to Egypt in 1840, and was employed on the West Coast of Africa to inquire into the administration of the British settlements. He lived in Lisbon from 1843 to 1846, acting as special correspondent to the *Morning Chronicle,* and afterwards went to Western Australia as Colonial Secretary. He ended his life as Secretary to the Local Fund Board in Dublin, where he died and was buried as a devout Roman Catholic ["Surgeons Who Were Never Surgeons", Sir D'Arcy Power, *The British Medical Journal,* October 5, 1929, page 628 (from knowledge obtained whilst editing Plarr's "Lives of the Fellows of the Royal College of Surgeons of England")].

Madden Mount, Western Australia, was named by Captain J. S. Roe in 1848 after Dr. R. R. Madden, then Colonial Secretary of Western Australia.

James Mitchell was appointed Assistant Surgeon, New South Wales, on June 11, 1823. He was the first resident medical officer-in-charge of Sydney Hospital, 1825-1837. He was the father of David Mitchell, who presented the Mitchell Library to Sydney.

Mitchell River, a sub-branch of the Namoi, was named by Allan Cunningham in May, 1827, "as a compliment to the medical gentleman to whom I am so much indebted for the valuable detail of barometrical observations he had taken for me in Sydney during my absence on this journey to the interior" (Cunningham's Journal in Ida Lee's "Early Explorers in Australia", page 552).

James Montgomery was surgeon of the *Bathurst,* Captain P. P. King's ship.

Montgomery Isles, near Camden Bay, Western Australia, were named by Captain Phillip P. King on August 16, 1821. Nine days previously Montgomery had been wounded in the back by a spear at Hanover Bay. (King's "Narrative of a Survey of the Intertropical and Western Coasts of Australia", Volume II, page 79.)

Baron Sir Ferdinand Jakob Heinrich von Mueller, K.C.M.G., M.D., Ph.D., F.R.S. (1825-1896), was the well known botanist. Von Mueller was not a medical man, his M.D. being an honorary one. He accompanied the Gregory brothers in their explorations in north Australia in 1855.

Mount Müller, in Western Australia, near 20° south and the South Australian border, was named by A. C. Gregory in 1856.

Mount Müller, near the Roper River, Northern Territory, was named by John McDouall Stuart in 1862.

Mueller Creek, a part of the Diamantina, near Mount Euro, and Mount Mueller (Mueller Range), a range of hills near it, were named by J. M'Kinlay on April 8, 1862.

County of Mueller, Queensland.

Mount Ferdinand, Ferdinand Creek, and Glen Ferdinand, in the Musgrave Ranges, were named by Ernest Giles. The two first named have been altered to Mount Warrabillinna and Ernabella Creek.

Sir Charles Nicholson, Bart., M.D., M.A., D.C.L., LL.D. (1808-1903), came to Australia in 1834. He was speaker three times of the Legislative Council of New South Wales. He was one of the founders of the University of Sydney and its chancellor in 1854. In 1859 he was speaker of the first Legislative Council in Queensland. An account of him is given by Mr. R. A. Dallen in the *Journal and Proceedings of the Royal Australian Historical Society,* Volume XIX, Part IV, 1933, page 213.

Mount Nicholson, near the Expedition Range, between the Dawson and Comet Rivers, Queensland,

was named by Leichhardt in November, 1844, "in honour of Dr. Charles Nicholson, who first introduced into the Legislative Council of New South Wales the subject of an overland expedition to Port Essington". A sketch is given of the mountain. (Leichhardt's "Journal of an Overland Expedition in Australia from Moreton Bay to Port Essington", 1847, page 51.)

William Alleyne Nicholson, of Bristol. Nicholson River, a branch of the Gregory River, which runs into the head of the Gulf of Carpentaria, was named by Leichhardt on August 20, 1845, after "Dr. William Alleyne Nicholson, of Bristol, whose generous friendship has not only enabled me to devote my time to the study of the natural sciences, but to come out to Australia" (Dr. Ludwig Leichhardt, *loco citato*, page 370).

Robert Peel was registered in South Australia in 1864; he was a licentiate of the King and Queen's College of Physicians, Ireland (1879). Dr. A. A. Lendon believes that he was Dr. Cawley's predecessor and later partner in a North Terrace practice, occupying a cottage now replaced by the Queen Adelaide Club, and adds that he left Adelaide before 1883, probably thus taking his Irish qualification in 1879. He had Melbourne as his address in 1901, and is remembered by Canon Poole as a charming singer when the Canon paid occasional visits to Mount Gambier.

Peel's Wells, Northern Territory, were named after him when he accompanied Goyder's party to Port Darwin in 1869.

Robert Purdie was surgeon's assistant on Flinders's ship, the *Investigator*.

Purdie's Isles, in Nuyt's Archipelago, Great Australian Bight, were named by Flinders in February, 1802, with other isles, "after young officers of the *Investigator*".

Dr. Purdie, of Edinburgh. Purdie's Ponds, Northern Territory, on the overland telegraph line, were named by McDouall Stuart, June 11, 1866, after "Dr. Purdie, of Edinburgh".

Dr. Ramsay owned Dobroyde Estate, near Sydney, about 1860. His son became Curator of the Australian Museum, Sydney.

Ramsay's Bush, near Iron Cove, Sydney, is named after him.

William Redfern (1778(?)-1833) was sentenced to death by a naval court martial at the age of nineteen, when surgeon's first mate in His Majesty's Ship *Standard*, for being implicated in the mutiny at the Nore in 1797, his offence being that he had verbally advised the leaders of the mutiny to be more united amongst themselves. The sentence was afterwards reduced to that of transportation. On his arrival in Sydney he was sent to Norfolk Island. In 1802 Governor King granted him an absolute pardon. In 1805 he was appointed an assistant surgeon in Sydney. He seems to have incurred unjustly the personal and rancorous hostility of Mr. Commissioner Bigge, which was probably the reason why he did not receive the appointment of principal medical officer. He was highly thought of by Governor Macquarie, and was frequently invited to his table, but the junior officers of the 46th Regiment refused to sit at table with him on account of his having been an emancipist. Professor G. A. Wood says that he became "the best doctor in Sydney, a big and successful farmer, and a man of high intellectual ability".

Redfern, the suburb of Sydney, is named after him.

Frederick Emil Renner, M.D. (Jena), 1847, was in Adelaide in the early fifties. He was the first practising physician in the Wentworth district (1857-1870). He returned to Adelaide in 1870, and was Government Medical Officer to the Overland Telegraph Parties (1871-1872). A photograph of him appears in "Exploring in the Seventies", by Alfred Giles (W. K. Thomas, *The Register*, 1926 or 1927, opposite page 48).

Renner Springs, between Tennant Creek and Powell Creek, is called after him. (Spencer and Gillen, "Across Australia".)

Dr. Shillinglaw, F.R.C.S., of Melbourne. Mount Shillinglaw, near Newcastle Waters, Northern Territory, was named by John McDouall Stuart on June 21, 1861, "after —. Shillinglaw, Esqr., F.R.C.S., of Melbourne".

Sir James Edward Smith, Kt. (1759-1828), was the famous botanist. In 1783 he studied under John Hunter, and in the same year bought the library, manuscripts, herbarium *et cetera* of Linnaeus. He founded the Linnean Society in 1788. He was author of "Flora Britannica", "The English Flora" and many other works.

Smith's Plains, on the lower Lachlan, New South Wales, were named after him.

Mr. Oxley named Smith's Plains in honour of Sir James Edward Smith, Kt., botanist and physician and author of several most valuable works as well on the botany of Australia as of countries less remote (Cunningham's Journal, June 25, 1817, in Lee's "Early Explorers in Australia", page 245).

Sir Edward Charles Stirling, C.M.G., F.R.S., M.A., M.D., F.R.C.S. (1848-1919), was professor of Physiology in the University of Adelaide.

Stirling Peak, Western Australia, was named after him (on the authority of the Surveyor-General of Western Australia).

Taillefer was surgeon of the French vessel *Naturaliste*.

Taillefer Prong, Western Australia, was named by Baudin's French expedition of 1801-1803.

Dr. Tate was botanist to William Hann's exploration party to Cape York, Queensland, in 1872. At Tate's request, Hann named Andrews Range after his friend Andrews, who was second mate on the *Maria*. This vessel, while making for New Guinea with an exploration party, had been wrecked in February, 1872, on the Great Barrier Reef, and Andrews had been drowned. Dr. Tate was on board

the *Maria* when she was wrecked. Dr. Tate was presumably a medical man.

Tate River, Queensland, was named by Hann.

CHARLES THROSBY, formerly a ship's surgeon, arrived in Sydney on June 13, 1802. He settled in the Wingiecarribee district in 1817 or 1818. With Hamilton Hume he explored towards the Wollondilly in 1817. His nephew Charles was the ancestor of the present Throsbys.

Throsby Park, New South Wales, was named in 1820.

GAVIN TURNBULL was a surgeon in the Indian Army.

Mount Turnbull, north of Central Mount Stuart, Central Australia, was named on May 3, 1860, by John McDouall Stuart. In his published journals Turnbull is referred to as "the late".

DR. VIGORS was an early Western Australian medical man.

Vigors Mount, Western Australia, was named by F. T. Gregory in 1858.

RUDOLPH VIRCHOW was the famous German pathologist.

Virchow Mount, Western Australia. Mr. J. C. Cannes, the Surveyor-General of Western Australia, states that (Sir) John Forrest in 1880 named this mountain after "Dr. Virchow, the Berlin throat specialist, who attended Frederick II, Emperor of Germany". It was not, however, till 1887 that Sir Morell Mackenzie was called in consultation to the then Crown Prince. The material that he removed at the first operation was handed for examination to Rudolph Virchow, the pathologist. Unless a mistake has been given in the date of naming, Sir John Forrest must have bestowed the honour for some other reason than the general interest created by the Royal illness. Virchow was, of course, already famous in 1880 as a pathologist, but would Sir John know him as such?

DR. WALKER was surgeon (and naturalist ?) on Captain (afterwards Sir George) Grey's expeditions to the north-west of Western Australia (Hanover Bay) in 1837 and 1838, and to Shark Bay in 1839. Dr. Walker was the first of the party left behind at Water Peak to arrive in Perth after Grey's disastrous journey on foot from Shark Bay.

Walker Valley, Hanover Bay, north-west of Western Australia, was named by Captain Grey in 1839 because of Walker's success in finding a road from Hanover Bay to adjacent country.

Walker Mount, Western Australia, was named by H. M. Lefroy in 1863.

JOHN WHITE (1750-1832) was Principal Surgeon to the Settlement of Botany Bay and Port Jackson from October, 1786, to December, 1794.

White's Creek runs into Parramatta River, one boundary of a grant to him in the Petersham district.

BRAIDWOOD WILSON, R.N. (1792(?)-1838(?)). Braidwood, a town in New South Wales, where Dr. Wilson settled in 1839, the town being laid out in 1840, was named after him.

W. H. WOLLASTON (presumably William Hyde) was born in the eighteenth century. He was noted for his scientific pursuits. I do not know whether W. H. Wollaston was a medical man or not.

Wollaston Island, near York Sound, north-west coast of Western Australia, was named by Commander P. P. King on September 7, 1820, after Dr. W. H. Wollaston.

DR. WOODFORDE was a well known medical man in Adelaide in the fifties and sixties.

Woodforde Creek in Central Australia, about ninety miles north of Alice Springs, was named by John McDouall Stuart on April 4, 1861. Dr. Woodforde's son John was a member of the party. There is now also a Woodforde Well on the creek.

Acknowledgements.

I would like to express my indebtedness to Dr. A. A. Lendon, of Adelaide, to Mr. F. I. Bray, of the State Archives Board, Perth, to the Surveyors-General of New South Wales, Queensland, South Australia, and Western Australia, and to the Secretary of the Department of the Interior, Canberra, for much valuable information and assistance.

A METHOD OF FEEDING CONCENTRATED MILK MIXTURES TO INFANTS.

By KATE CAMPBELL, M.D. (Melbourne),
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IT is a matter of general observation that infants do not as a rule manage well whole unmodified cow's milk. It has therefore been the custom to dilute the milk with water in varying strengths. The obvious result of dilution of cow's milk is to render the nutritive value of the mixture less, in amount depending on the degree of dilution. There are two means by which the food value of the mixture can be increased: by the addition of suitable carbohydrate and by the addition of fat. The addition of carbohydrate is universal as a modification of milk for the use of infants, and is well tolerated. The addition of fat is a more difficult matter, depending on the individual baby and also on the climate. In cold weather and in colder climates the fat appears to be better tolerated than in hotter seasons and climates. In Melbourne, I think, most physicians agree that fat dyspepsia is considerably more frequently met with than are other types of indigestion. Many babies undoubtedly manage quite considerable additions of fat, but when fatty tolerance is limited, when the climate is unsuitable, or when the infant cannot be kept under adequate supervision, addition of fat is undesirable. Another difficulty which sometimes arises is that the fat content of cow's milk is a variable and often

unknown quantity. It is difficult to obtain a milk with a standardized fat content, and most specimens of milk tend to exceed, quite definitely, the Victorian legal requirement of 3·5% butter fat. Fat is a concentrated food, and its addition to mixtures of milk, water and sugar is very useful in raising the nutritive value of such mixtures. If, however, conditions prevail which render the addition of fat impossible, we are left with mixtures of milk, water and sugar. Such feedings, while in general easy of digestion, suffer from the disability of a low nutritive or caloric value. A mixture of milk and water, equal parts, with carbohydrate added up to 7%, gives approximately 14·8 calories to the ounce. Breast milk gives 20 calories to the ounce, so that to give the same nutritive value, the milk and water feeding must be one-third as large again as the feeding of breast milk. All the usual milk, water and sugar mixtures suffer from this disability of relatively low nutritive value.

Roughly speaking, with the addition of carbohydrate to between 6% and 7% the following is the caloric value per ounce of the different strength feedings usually employed:

$Milk_1$ $water_3$ = 11 calories.
$Milk_1$ $water_2$ = 12 calories.
$Milk_2$ $water_3$ = 13 calories.
$Milk_1$ $water_1$ = 14 calories.
$Milk_2$ $water_2$ = 15 calories.
$Milk_2$ $water_1$ = 16 calories.
$Milk_3$ $water_1$ = 17 calories.

The administration of feedings of these relatively low caloric values presents the difficulty that large feedings are required to satisfy the child's nutritive requirements. For example, an infant of five to six weeks, weighing eight and a half pounds, if fed on a milk and water equal parts mixture, requires 30 ounces per day to give it sufficient nourishment. This means six-ounce feedings every four hours, whereas with breast milk it would require 21 ounces per day, roughly four-ounce feedings every four hours. With dilute mixtures some infants with small appetites will not take big enough feedings. Most infants, however, will take the amounts necessary, and there is then more tendency to regurgitation and vomiting than when smaller feedings can be employed. It frequently necessitates three-hourly feeds, and the infant passes very large amounts of urine and tends to be always wet. The babies are also less comfortable than infants on the smaller feedings.

In an endeavour to overcome these disabilities it occurred to me that, perhaps, the large amounts of water present in the feedings might not be really necessary for digestive purposes and that it was possible that the amounts of carbohydrate, fat and protein taken per day might be more important than their percentage composition in the feed. Consequently the babies were given feedings which contained the same amount of milk and sugar as in the orthodox feedings, but the amount of water added was sufficient only to give a caloric value of 20 to the ounce. These feedings were known as "the concentrated mixtures". By using a 20-calorie-ounce mixture we satisfy the child's nutritive needs with a feeding which also yields enough water for the child's fluid requirements. The result of these feedings was very satisfactory, the infants digesting the mixtures well and gaining very satisfactorily. The stools were normal. In many we noticed an improvement in appetite, and also less tendency to scalded buttocks consequent on the diminution of the too free urinary secretion. Four-hourly feedings became possible in almost every case.

Such formulæ have been fed to all the artificially fed and complemented infants in residence at the Victorian Baby Health Centres Mothercraft Home since August, 1933, with good results.

These concentrated feedings have been found especially useful in those new-born infants who require to be either complemented or completely artificially fed. These infants take, as a rule, only small feeds, and the concentrated formulæ allow the baby to ingest greater amounts of nutriment per day with a consequent improvement in its nutrition.

In the case of difficult feeders concentrated mixtures fulfil a definite need.

In the feeding of premature infants where breast milk cannot be obtained, these mixtures have also been employed. The number of premature infants so treated up to date has not been large enough to make an authoritative statement, but the results have been very encouraging.

In dyspeptic infants it has been found that they progress more favourably on these formulæ and make more rapid convalescence than on the more dilute mixtures. In some cases so marked an improvement has been observed when the feeding has been changed to the concentrated mixture that it would almost suggest that the excess water had previously hampered digestion, perhaps by excessive dilution of the digestive ferments, although this is purely speculative.

These feedings have a high percentage composition, and in Table I are the compositions of the usual and of the concentrated mixtures. The amounts of carbohydrates added are those given in Dr. Vera Scantlebury's "Guide to Infant Feeding", which is used as a standard in Victoria.

Table I.

Strength.		Usual Mixtures.			"Concentrated Mixtures."		
		Carbohydrates.	Fats.	Proteins.	Carbohydrates.	Fats.	Proteins.
M.[1]	W.[2]						
1	3	7·0	0·9	0·9	12·1	1·5	1·5
1	2	7·0	1·1	1·1	11·2	1·9	1·9
2	3	6·9	1·4	1·4	10·2	2·1	2·1
1	1	6·8	1·7	1·7	9·4	2·4	2·4
3	2	6·7	2·1	2·1	8·4	2·7	2·7
2	1	6·6	2·3	2·3	8·0	2·9	2·9
3	1	6·5	2·6	2·6	7·4	3·0	3·0

[1]Milk. [2]Water.

Below are given the amounts necessary for making 20 ounces of each of the various strengths of the concentrated feedings. The milk and water

are simmered together in a double boiler for ten to twenty minutes. Any loss in bulk due to the heating is made up by the addition of the appropriate amount of boiled water.

Concentrated Milk$_1$ Water$_3$:
Milk 8¾ ounces.
Lactose 5 tablespoons 1¾ teaspoons.
Water to 20 ounces.

Concentrated Milk$_1$ Water$_2$:
Milk 10½ ounces.
Lactose 4 tablespoons 3 teaspoons.
Water to 20 ounces.

Concentrated Milk$_2$ Water$_3$:
Milk 12 ounces.
Lactose 4 tablespoons.
Water to 20 ounces.

Concentrated Milk$_1$ Water$_1$:
Milk 14 ounces.
Lactose 3 tablespoons 1½ teaspoons.
Water to 20 ounces.

Concentrated Milk$_2$ Water$_2$:
Milk 15½ ounces.
Lactose 2 tablespoons 2 teaspoons.
Water to 20 ounces.

Concentrated Milk$_2$ Water$_1$:
Milk 16½ ounces.
Lactose 2 tablespoons ½ teaspoon.
Water to 20 ounces.

Concentrated Milk$_3$ Water$_1$:
Milk 17½ ounces.
Lactose 1 tablespoon 3 teaspoons.
Water to 20 ounces.

In measuring these amounts standard measures are employed. The sugar is measured in tightly packed and levelled spoons.

I have employed the same methods in using Nestlé's condensed milk. The usual strength of 1 in 8 by volume gives a 16·4 calorie-ounce mixture. A concentration of 1 in 6½ gives a 20 calorie-ounce mixture with a percentage composition of carbohydrates 11·2, fat 1·9, protein 1·9. This is very well tolerated by miserable dyspeptic infants.

Method of Calculating the Mixture.

The method of calculating the mixture was as follows. Suppose an infant would need 30 ounces of a milk and water equal parts mixture. The usual formula would be 15 ounces of milk, 15 ounces of water, 3 tablespoons 2 teaspoons of lactose. The caloric value of the feeding would be 30 × 14·8 = 444 calories. Using a 20 calorie-ounce mixture the bulk of the feeding would be 444 ÷ 20, that is 22 ounces. This means the addition of 7 ounces of water, so that the formula for what we may term a concentrated equal parts mixture would be 15 ounces of milk, 7 ounces of water, 3 tablespoons 2 teaspoons of lactose. At first sight this looks like an orthodox milk$_2$ water$_1$ mixture, but it is really not so, the sugar content being considerably higher than prevails in the customary milk$_2$ water$_1$ mixture.

If it is desired to calculate the amounts of water necessary to make up amounts of the concentrated mixtures equal to 20 ounces of the various strengths of the usual mixtures, an easy method is as follows. Take the amounts of milk and sugar given to make up 20 ounces of the usual mixture. The figure of the caloric value per ounce of the usual mixture gives the total bulk of the concentrated mixture yielding the same nutritive value. Therefore, the difference between this figure and the amount of milk given is the amount of water which must be added. For example, for 20 ounces of milk$_1$ water$_1$ the usual formula would be ten ounces of milk, ten ounces of water, 2 tablespoons 1½ teaspoons of lactose. The caloric value per ounce of this mixture is 14·3. For the concentrated mixture of equivalent value we would require 10 ounces of milk, 2 tablespoons 1½ teaspoons of lactose, and water added to make the bulk of the feeding up to 14·3 ounces, that is, 4⅓ ounces of water.

Practical Application of These Feedings.

In feeding any particular infant on these concentrated mixtures one first calculates the theoretical caloric requirements at so many calories per pound of body weight. (The standard adopted is 50 calories per pound of body weight for the first three months, 47 for the fourth month, 45 for the fifth, 44 for the sixth, 43 for the seventh, and 42 for the rest of the first year.) The theoretical caloric requirements divided by twenty give the number of ounces of the mixture required per day, and the mixture suitable for the age of the child is selected. From the nutritive point of view, all the mixtures have the same value, but it is customary to put the infant on the stronger mixtures as it grows older. The concentrated milk$_1$ water$_3$ and milk$_1$ water$_2$ mixtures are not used as permanent feedings, being used as introductory feedings, as in the case of new-born infants or those being weaned.

The manner in which the infants manage these mixtures has impressed upon me the viewpoint that one should not regard the amount of water in the feeding mainly from the digestive standpoint. With marked reduction in the amount of water the feedings were managed even better, so that the rôle of water as a digestive agent has, I think, been over-stressed. Rather we should look upon the amount of water present more as a means of satisfying the infant's fluid requirement, which is usually gauged at two and a half ounces per pound of body weight per day.

As compared with breast milk, these mixtures are lower in fat and higher in carbohydrate and protein.

They correspond roughly with the "majority group" of Grover F. Powers,[1] who divided foods for infants into groups depending on the proportion of calories yielded by each element. He found that most of the empirically used artificial foods fell into several well defined groups. The "majority group" were those which had been found in general to be most successful. In this group carbohydrates yielded 60% to 65% of the total calories, protein 10% to 20%, and fat 15% to 30%.

In general, as compared with the majority group, these concentrated mixtures yield about the same proportion of calories from the protein but rather less from the carbohydrate and rather more from the fat.

The suitability of a feeding mixture cannot be judged by theoretical considerations only, the guide being, of course, the progress and well-being of the infant. Over a period of months in the same infants these mixtures have survived this test.

A good deal is heard about over-feeding in infants, but it has been my experience to meet with very many cases of under-nutrition, as compared with a very limited number of cases of over-feeding, and I am sure that under-feeding is a very common trouble, much more common than dyspepsia, for which under-feeding is frequently mistaken. In the majority of cases it is due to too weak mixtures being employed, the babies being given too small a feeding, or else being incapable of taking sufficient. If more concentrated mixtures are employed, the risk of under-feeding becomes very much less.

As would be expected, infants on these more concentrated mixtures tend to be more constipated than when on more dilute feeding, unless suitable training in holding out *et cetera* is practised.

Acknowledgements.

In conclusion, it is with pleasure that I express my indebtedness to Matron Kerville and Sister Sage, of the Victorian Baby Health Centres Association Mothercraft Home, Sister Hawkins and Sister McRae, of the Queen Victoria Hospital, and Sister White, of the Women's Hospital, for their cooperation and observation in testing these feedings on the infants in their respective charge, and take this opportunity of expressing my appreciation of their support.

Reference.

(1) Grover F. Powers: "Comparison and Interpretation on a Caloric Basis of Milk Mixtures Used in Infant Feeding", *American Journal of Diseases of Children*, Volume XXX, October, 1925, page 453.

THE TREATMENT OF DORSAL DISPLACEMENT OF THE LITTLE TOE.

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Dorsal displacement of the little toe is a very troublesome affection. It is usually so painful that relief is sought, and this is usually met by amputation of the toe. This is a disfiguring operation and relief may be obtained by another simple procedure.

The deformity is due to a contracture involving the skin and subcutaneous tissues over the head of the fifth metatarsal bone. This is shown by the manner in which the toe may be made to resume its normal position on cutting the skin and subcutaneous tissues transversely to the head of the bone. The extensor tendons are also shortened and help to retain the toe in the deformed position.

Treatment.

The deformity is best dealt with by a pedicled skin graft and by tenotomy of the tendon of the *extensor communis longus* and, if necessary, of the *extensor brevis*. The skin graft corrects the defor-

Figure I.

Figure II.

mity by filling in the gap left after correction of the deformity and is carried out as shown in the diagrams. The tenotomy corrects the shortening of the tendons of the dorsiflexing muscles.

VIRULENCE TESTING IN DIPHTHERIA.

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and

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In 1884 Löffler discovered the causative organism of diphtheria; later it was found that diphtheria bacilli morphologically identical and giving the same fermentation reactions were divisible into groups. One group contained virulent organisms and the other avirulent organisms.

The term virulence in its general use is a loose one and may be taken to mean invasiveness or toxicity or both, but, when used in respect to diphtheria, toxicity of the organism is implied. It is clearly desirable to know whether bacilli isolated from a throat culture and morphologically resembling diphtheria bacilli are virulent or avirulent. Without this knowledge proper control of the carrier cannot be attained. The practical importance of this is to be seen in the possible prolonged isolation of a carrier of avirulent organisms, and also that many schemes of control of outbreaks of diphtheria in institutions are dependent to some extent on virulence testing. Although many articles on diphtheria have appeared in Australian literature, there has been comparatively little on this aspect of the subject.

To test the virulence of a diphtheria culture, animal inoculation is necessary, and the animal in general use for this purpose is the guinea-pig. That

the susceptibilities of man and guinea-pig are sufficiently alike for this purpose is accepted, and the authors of the Medical Research Council's publication on diphtheria[1] sum up the evidence on this point as follows:

> In our opinion, therefore, the evidence clearly supports the view that a strain non-virulent to guinea-pigs is non-virulent to man, and that a strain virulent to guinea-pigs, in whatever degree, is virulent to man, but not necessarily in the same degree. Thus information derived from guinea-pigs may be applied safely to the study of diphtheria in man.

Two methods, with several variations, are used in this test. In each, two guinea-pigs are needed, one of which is protected by the previous inoculation of diphtheria antitoxin. In the first method the culture or filtrate is inoculated subcutaneously, in the second intracutaneously. The latter has the advantage of economy in that several different cultures may be tested in the same two animals.

A modification of the intracutaneous test used by Havens and Powell[2] in 1922, and later by Force and Beattie[3] and others, is the use of a suspension of the original diagnostic culture, generally termed the "field culture".

The method used as a routine in the following series was the "field culture" intracutaneous method. The reason for this choice was economy in guinea-pigs and in time. The cultures tested came from cases at Canberra and this occasioned a delay of approximately twenty-four hours; and, therefore, consideration of time was of importance.

The results obtained in other investigations show a fairly close relation between pure culture and field culture methods. In both methods there is a difficulty. The pure culture method has the disadvantage that the test culture is one of many colonies, and it is possible that the plate contains both virulent and avirulent colonies and that one of the latter may be selected. Attention has been drawn to a weakness in the field culture method by Okell and Parish,[4] who found: "Impure primary ("field") cultures gave reliable results (94%) when more than one-third of the organisms seen in the smear made from the overnight culture were morphologically *C. diphtheriæ*. If fewer *C. diphtheriæ*, the primary culture gave uncertain results."

The technique and results of this investigation were as follows:

Technique.

All cultures on arrival at this school were placed in the incubator at 37° C. overnight. The following morning a representative sample was taken from the inspissated serum culture and smears were prepared. The smears were stained by toluidine blue and thoroughly examined under the $^1/_{12}$ oil immersion lens. The results were recorded according to a definite system. In those cases in which organisms resembling *Corynebacterium diphtheriæ* morphologically were the predominating organisms in the microscopic field, the result was recorded as ++. In cases in which the diphtheria-like organisms were approximately one-quarter to one-third of the organisms in the field, the result was marked +. When extremely few organisms resembling *Corynebacterium diphtheriæ* were found, the word "occasional" or "scattered" was used. With the exception of some thirty cultures at the commencement of these examinations, virulence tests were performed on all microscopically positive cultures. The method employed for virulence testing was the intracutaneous method, "field" cultures being used.

The growth from an eighteen to a twenty-four hour culture on Löffler's inspissated serum medium was emulsified in sterile saline solution and transferred to a sterile test tube. The suspension was then further diluted to approximately 500 million to 1,000 million organisms per cubic centimetre, as judged by the opacity method. The stronger suspension was used when the diphtheria bacilli appeared to be scanty in the stained smears. Two white-haired guinea-pigs were used in each test. One animal was given 500 units of diphtheria antitoxin by subcutaneous inoculation twenty-four hours previous to the test. This animal acted as a control. The fur was removed from the abdomen of the animals by clipping and shaving or by depilation. Of each suspension to be tested 0·2 cubic centimetre was injected into corresponding skin areas of the two animals. The injections were intradermal and at least 2·5 centimetres (one inch) apart. One cubic centimetre "Vim" tuberculin syringes were used with 26 gauge needles with short bevels. Four to six cultures were tested on each guinea-pig. Readings were taken twenty-four, forty-eight, and seventy-two hours after the injections were made. The control animals showed in general only slight traumatic effects of the needle puncture. The test animal with sufficient virulent diphtheria bacilli showed a reddish swelling, which became more marked at each reading, terminating in a slight necrosis.

Results.

The results obtained are grouped in two series, A and B. In Group A the clinical condition of the individual from whom the swab was taken is known.

Group A: Cultures examined between March 10, 1931, and May 14, 1932 ..	1,568
Group B: Cultures examined between May 14, 1932 and November 30, 1932	286
Total	1,854
Group A: Virulence tests	512
Group B: Virulence tests	106
Total	618

Group A.[1]

Total number of cultures examined	1,568
Total number of virulence tests	512 (32·0%)

[1] In this statement of results M.E. represents microscopic examination; the letters V.T.R. represent virulence test result.

Relation between result of microscopic examination and virulence test:		
(1) M.E., ++; V.T.R., +ve	342	(87·4%)
M.E., ++; V.T.R., −ve	49	(12·6%)
M.E., ++; V.T.R., ?	0	
Total	391	
(2) M.E., +; V.T.R., +ve	35	(38·5%)
M.E., +; V.T.R., −ve	55	(60·4%)
M.E., +; V.T.R., ?	1	
Total	91	
(3) M.E., occasional bacilli only; V.T.R., +ve	1	(3·6%)
M.E., occasional bacilli only; V.T.R., −ve	27	(96·4%)
M.E., occasional bacilli only; V.T.R., ?	0	
Total	28	
(4) M.E., doubtful; V.T.R., −ve	2	
(5) M.E., +; no V.T.	30	

(These represent some cases at the commencement of the investigation, chiefly cases in which virulence tests were not repeated with each culture.)

(6) Total number of individuals swabbed in Group A	628	
(a) Total number of clinical cases of diphtheria in this group	140	(22·3%)
(b) Total number of contacts of diphtheria cases,	467	(74·4%)
(c) Total number with insufficient data to classify, and miscellaneous (tonsillitis, Schick test *et cetera*),	21	(3·3%)
(6A) Results of examination of clinical cases of diphtheria, showing relation between microscopic examination and virulence test:		
M.E., +; V.T.R., +ve	79	(56·4%)
M.E., +; V.T.R., −ve	18	(12·9%)
M.E., +; V.T.R., ?	1	(0·7%)
M.E., doubtful; V.T.R., −ve	1	(0·7%)
M.E., −ve	41	(29·3%)
Total	140	
(6B) Results of examination of contacts of diphtheria cases, showing relation between microscopical examination and virulence test:		
M.E., +; V.T.R., +ve	29	(6·2%)
M.E., +; V.T.R., −ve	57	(12·2%)
M.E., ?; V.T.R., −ve	1	(0·2%)
M.E., −ve	380	(81·4%)
Total	467	
(6C) Results of examination of cases with insufficient data to classify, and miscellaneous:		
M.E., +; V.T.R., +ve	0	
M.E., +; V.T.R., −ve	1	
M.E., −ve	21	
Total	22	

Group B.

(May 14, 1932, to November 30, 1932.)

Total number of cultures examined	286	
Total number of virulence tests	106	(37·0%)
Relation between result of microscopical examination and virulence test:		
(1) M.E., ++; V.T.R., +ve	73	(83·9%)
M.E., ++; V.T.R., −ve	14	(16·1%)
M.E., ++; V.T.R., ?	0	
Total	87	
(2) M.E., +; V.T.R., +ve	6	(46·2%)
M.E., +; V.T.R., −ve	7	(53·8%)
M.E., +; V.T.R., ?	0	
Total	13	
(3) M.E., occasional bacilli only; V.T.R., +ve	0	
M.E., occasional bacilli only; V.T.R., −ve	5	(100%)
M.E., occasional bacilli only; V.T.R., ?	0	
Total	5	
(4) M.E., doubtful; V.T.R., −ve	1	

A consideration of these results clearly indicates an association between the number of diphtheria bacilli in the microscopic field of the smear preparation and the result of the virulence test, and a statistical comment on this is now given.

Virulence and Microscopical Picture.

Is there any association between these two attributes? First we must discard doubtful cases. This leaves us with 509 virulence tests. The results of virulence tests are divided into two classes, which may be written V+ and V−. Microscopic appearances are divided into three classes, ++, +, and occasional bacilli, which may be distinguished by the signs M++, M+, and M_{occ}. We may divide our virulence test results into two samples, that is, V+ and V−. Now if these distributions are significantly different as regards their grouping into M++, M+ and M_{occ}, then microscopic appearances and virulence tests have a definite association.

We can best test for association by the Chi^2 test (Table I).

Table I.

Section.	Class.	M++.	M+.	M_{occ}.	Result.	Total.
V+	(1)	342	35	1	f	378 = N
V−	(2)	49	55	27	f′	131 = N′
(1) + (2)	(3)	391	90	28	f + f′	509
ω/N	(4)	0·9048	0·0926	0·0026	f/N	1·0000
ω'/N'	(5)	0·2704	0·4198	0·2062	f'/N'	1·0000
(4) − (5)	(6)	0·5208	0·3272	0·2306	$f/N - f'/N'$	
(6)²	(7)	0·281749	0·107060	0·041452	$(f/N - f'/N')^2$	
ω/ω	(8)	0·000720586	0·001180297	0·001480464	$\frac{(f/N - f'/N')^2}{f + f'}$	0·003381347

$$Chi^2 = NN' \times (8)$$
$$= 378 \times 131 \times 0{\cdot}003381347$$
$$= 49518 \times 0{\cdot}003381347$$
$$= 167{\cdot}4375$$

Now $n = 2 + 1 = 3$
For $n = 3$ and $Chi^2 = 167$
P is less than 0·000001

Therefore there is less than one chance in 1,000,000 that these results are due to chance, and so we may say that with positive microscopic slides virulence is closely associated with the number of bacteria; the more bacteria, the more chance of a positive result to a virulence test. It is regretted that virulence tests were not done on the negative microscopic slides as well. Consumption of time and the number of animals required forbade this. The result is, however, the qualification that the association holds only for positive microscopic slides, that is, we cannot say that virulence varies directly with the number of bacteria present on the slide. This only holds true for positive slides, negative slides not being included for statistical treatment.

It is to be noted that the association between the number of bacteria seen in the microscopic field and positive results to virulence tests is really closer than indicated above, for when but few bacteria were seen on examination the opacity of the suspension inoculated into the pig was intentionally increased. Such suspension would probably tend to give an increased number of positive reactions.

Treating the second series in the same way we get:

$Chi^2 = 24{\cdot}6$ and $n = 3$
That is, P = 0·000005

That is, only one chance in 200,000 that such different arrangements could be due to chance.

Combining our two groups, we get $Chi^2 = 191{\cdot}8$, $n = 3$, which gives a probability of less than one in a million that the association of the number of bacilli on a microscopic slide with a + virulence test is due to chance.

Certain other points of interest came under notice, and were investigated as far as circumstances permitted.

1. A comparison of the virulence of the Canberra strain and the Park 8 strain was made by intracutaneous test on guinea-pigs. The Canberra strain tested was a pure culture isolated from D.K., and the Park 8 strain was isolated by Park and Williams in 1895, and is the standard strain used in the preparation of diphtheria toxin.

From the above the following eight suspensions were prepared with sterile normal saline solution.

400 million per cubic centimetre.
300 million per cubic centimetre.
200 million per cubic centimetre.
100 million per cubic centimetre.
50 million per cubic centimetre.
20 million per cubic centimetre.
10 million per cubic centimetre.

Intracutaneous tests were made, all of the above suspensions being used. On final reading necrotic areas were present at the sites of inoculation in every case, the intensity of reaction being proportional to the strength of the suspension. The reactions from the Park 8 strain appeared slightly more marked. The control guinea-pig showed no reaction.

2. As in experiment 1, the size of the guinea-pigs used did not permit of all tests being performed on the same animal; a somewhat similar test was performed, a pure white rabbit being used. The method advocated by Mader and Halpern[5] was used. The Canberra strain tested was a pure culture obtained from J.M. The following suspensions of Park 8 strain and J.M. strain were made:

200 million per cubic centimetre.
100 million per cubic centimetre.
50 million per cubic centimetre.
25 million per cubic centimetre.
10 million per cubic centimetre.

Intracutaneous inoculations were made on the rabbit over the depilated dorsal area, both strains being inoculated into the same animal. On the second day positive reactions were seen as follows:

Park 8 strain, 200, 100, and 50 million per cubic centimetre.
J. M. strain, 200 and 100 million per cubic centimetre.

The reactions were intensified, but not altered, on the third day. The result of this experiment tended to confirm the result of experiment 1—that Park 8 strain showed slightly more marked reaction.

3. As it was noted in experiments 1 and 2 that suspensions of pure cultures of Park 8 strain and Canberra strains gave positive results to virulence tests in dilutions of lower strength than appeared (by microscopic field examination) in the case of field cultures of Canberra strains, it was decided to test whether the addition of staphylococci would affect in any way the result of intracutaneous tests.

A suspension of 400 million per cubic centimetre was made in saline solution from a twenty-four hour growth on Löffler's inspissated serum medium of Park 8 strain. This suspension was diluted with saline as follows:

200 million per cubic centimetre.
100 million per cubic centimetre.
50 million per cubic centimetre.
20 million per cubic centimetre.

To each dilution an equal quantity of a 2,000 million per cubic centimetre suspension of *Staphylococcus aureus* was added. The result was that the above suspensions of Park 8 strain were changed to 100 million per cubic centimetre, 50 million per cubic centimetre, 25 million per cubic centimetre, and 10 million per cubic centimetre; and each dilution contained 1,000 million staphylococci per cubic centimetre.

A Canberra strain (D.K.) was treated in exactly similar manner. Intracutaneous tests of each dilution were made on protected and unprotected guinea-pigs.

The final readings showed positive reactions on the unprotected guinea-pig in all dilutions, the intensity of reactions being proportional to the strength of the suspension. The addition of staphylococci did not appear to affect the reaction noticeably.

4. A test was made to determine the existence of *Corynebacterium diphtheriæ gravis* and *mitis*. Anderson, Happold *et alii*[6] in 1931 reported the isolation of two principal forms of diphtheria bacilli from clinical cases. These were given subspecific names: (*a*) *Corynebacterium diphtheriæ gravis*, associated with severe toxic cases, and (*b*) *Corynebacterium diphtheriæ mitis*, associated with milder cases. A special tellurite chocolate medium was used for differentiation. Anderson's medium was prepared in strict accordance with his directions and forty positive Canberra cultures were plated out. In no case did the Canberra cultures show colonies resembling the descriptions and illustrations of *Corynebacterium diphtheriæ gravis*. Five pure cultures from different country districts in New South Wales were also tested with a similar result.

5. Some persistent carriers were noticed, two returning positive results for nine months. It is interesting that negative results in these cases were not obtained until after tonsillectomy was performed.

Comment.

In commenting on these results, emphasis may be placed upon certain aspects.

1. Although virulence testing is of the greatest importance in the control of diphtheria, as presenting the only means of final diagnosis of virulent organisms, yet the possibility of certain fallacies has to be borne in mind. A positive result to a virulence test by any recognized method can be relied upon to indicate the presence at that time of virulent *Corynebacterium diphtheriæ*. A negative result to a virulence test is not always conclusive. In the pure culture method there may be virulent and avirulent strains on the same place. In the field culture method the results show the association between the number of organisms in the microscopic field and the result of the virulence test. As previously stated, attention has been drawn to this point by Okell and Parish.[4] The number of cases tested in this investigation, and the statistical treatment of these figures add marked confirmation to this. From a practical aspect it would help in the interpretation of the results of virulence testing if workers in laboratories using the field culture method stated the relative frequency of diphtheria organisms on the microscopic preparation.

2. Comparative tests by intracutaneous inoculation appeared to indicate that virulence, as judged by this method, was slightly less in the Canberra strain than in the Park 8 strain.

3. No strains were found that resembled *Corynebacterium diphtheriæ gravis*.

Acknowledgements.

We are indebted to the Medical Officer of Health, Canberra, for the throat cultures tested and for the clinical particulars of the cases, and to Mr. K. J. Clinton, of the School of Public Health and Tropical Medicine, who very accurately performed the bulk of the technical work of this investigation.

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Reports of Cases.

HYDATID CYST OF THE BRAIN.

By T. Y. Nelson, M.B., Ch.M., F.R.A.C.S.,
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with Commentary by

Harold R. Dew,
Bosch Professor of Surgery, University of Sydney.

Because of its comparative rarity and because of the interesting nature of the clinical story and its investigation, the following case is thought worthy of record.

D.P., a well built boy of thirteen, was admitted to the Royal Alexandra Hospital for Children on February 17, 1933, complaining of headache and failing vision. He had first come under observation at the Royal North Shore Hospital of Sydney in August, 1932, when he complained of frontal headaches which had been present over a period of about five months, and some interference with vision for two months. During this period he had had several attacks of vomiting and had lost about 2·7 kilograms (six pounds) in weight. Examination of the eyes by Dr. Temple Smith revealed double papilloedema of approximately two diopters. No interference with the function of any of the other cranial nerves was found, and a general examination of his nervous system revealed nothing abnormal. X ray examination of the skull at this time showed no abnormality, but in October, 1932, the radiologist reported slight widening of the sagittal and upper part of the coronal sutures, and it was suggested that this was due to slight chronic rise of intracranial tension. The Casoni test and the hydatid complement fixation test both gave no reaction.

In February, 1933, when he was admitted to the Royal Alexandra Hospital for Children, Dr. Temple Smith reported two millimetres of swelling of both disks, but thought the pathological changes were less marked than earlier, there being less engorgement of the vessels and less swelling. The visual fields were examined by Dr. Gregg and showed no abnormality.

On February 28, 1933, on Professor Dew's advice, ventricular estimation was carried out by Deery's method. Briefly, the technique is as follows.

With the patient supine and shoulders resting on the table, the occiput is raised about 30 centimetres (twelve inches) above the level of the table and supported on a suitable head rest, leaving the occipital region free and

well elevated. The mid-line is marked on the skin by a line joining the nasion to the inion and a point is marked on each side, 2·5 centimetres lateral to the mid-line and 6·5 centimetres above the inion. This marks the centre of the trephine hole to be made. Under local anæsthesia a horizontal incision is made on each side and a button of bone removed, the dura incised and turned back and the cortex thus exposed. The ventricle is tapped on each side by inserting a brain needle parallel to the floor of the theatre. If the trephine holes are symmetrically placed and the pressure of and the amount of fluid obtained from each ventricle are recorded, valuable information may be obtained as to the relative sizes of the ventricles, the degree of internal hydrocephalus or of displacement of ventricles laterally. If a ventriculogram is required, the needles are left in position and the head is rotated gently from side to side until fluid ceases to flow, when the needles may be withdrawn, the incision closed and the necessary radiographs taken.

In this case the left side was punctured first and fluid under moderate pressure was encountered at a depth of one centimetre. In order to tap the right ventricle it was necessary to direct the needle somewhat laterally, and fluid under normal pressure was encountered at a depth of about four centimetres.

Without the introduction of air and X ray examination these findings suggested a large dilated left ventricle or a cystic collection with displacement of the right ventricle laterally. The radiograph showed, however, that the needle on the left side had entered a cyst which did not communicate with the ventricular system, although air had entered and filled the ventricular system through the needle on the right side. The diagnosis of hydatid cyst or cystic astrocytoma was then made and operation was advised.

On March 15 craniotomy was performed by Professor Dew, assisted by myself, under local anæsthesia preceded by "Sodium amytal", 0·72 gramme (12 grains). An osteoplastic flap was turned down in the left occipito-parietal region and the dura was reflected in the ordinary way. After ligation of a few cortical vessels behind the parieto-occipital sulcus, a needle was passed to relieve tension slightly, the cyst being struck at a depth of three centimetres. The cortex was then incised with the diathermy knife and the cyst entered from above. By careful packing and the use of a powerful sucker, all fluid was rapidly removed without any contamination of the field. The collapsed laminated membrane was then seen and was coaxed out of the cortical incision in its entirety. The cavity was then swabbed clean several times and the bottom was swabbed lightly with a pledget wet with Zenker's solution. The large cavity left after removal of the cyst was found to be bounded on its medial side by the ependymal lining of the ventricle. Owing to the close contact of the thin wall of the lateral ventricle, it was thought that the risk of using such a drastic fixature as formalin was contraindicated. After very careful toilet the cavity was filled with Ringer's solution at body temperature and the flap was closed in layers by Cushing's method of closely placed silk sutures. During the operation there was a marked fall in the blood pressure, and as a safeguard the boy was given a transfusion of 300 cubic centimetres (ten ounces) of citrated blood.

Convalescence was uneventful and examination eight months later showed that the optic disks had returned to normal, and that the patient was free of all symptoms.

Comment.

(H. R. Dew.)

This case has some interesting features. The patient is a little over the average age, but as regards latency the case is typical. The cyst was large, but did not involve either the optic radiation or the occipital visual cortex, so that there were no localizing signs. The presence of a cyst so close to the cortex, of such a size, and under such pressure immediately suggested the possibility of an hydatid cyst, although in the absence of response to any specific test certainty was impossible. The appearance in the radiogram of some trabeculation which is due to the comparative shadow cast by the crumpled laminated membrane is, of course, also typical and is similar to that sometimes given by a collapsed hydatid cyst of the lung. Examination of the fluid from the two punctures revealed, too, a relatively high salt content in the left side, but no scolices or hooklets, but it was therefore of some confirmatory value.

The close contact of the cyst wall with the thin ependymal wall of the lateral ventricle, which could be seen pulsating in the depths, is of importance, and it would not have been surprising if the puncture, as in the somewhat similar pulmonary cases, had, owing to sudden relief of support, precipitated rupture into the ventricle with a fatal result. This is worth bearing in mind in the case of similar deeply placed cysts.

Acknowledgements.

I am indebted to Dr. Temple Smith and Dr. R. B. Wade for permission to report this case.

Reviews.

OBSTETRICS.

"Practical Obstetrics" is the result of an endeavour to provide the "student with a concise text-book, the practitioner with a dependable guide and the specialist with an exposition of our personal views on current obstetric problems".[1]

The arrangement of the chapters is on standard lines, but there are included a chapter on obstetric jurisprudence and a section of referred reading.

The summary of the present state of knowledge of menstruation and its controlling hormones is a good one, and clear diagrams are included. The Zondek-Aschheim test is given fully in the chapter on the diagnosis of pregnancy, but it seems hardly necessary to give detailed tests relative to decreased sugar tolerance which the author admits are not very reliable.

The description of Hegar's sign is difficult to follow:

> The (lower uterine) segment may also be recognized by vaginal examination alone, with the thumb on the anterior and two fingers on the posterior surface of the enlarged organ, as well as by recto-vaginal examination with the index finger in the rectum and the thumb in the anterior fornix.

It is unwise and unnecessary to advise routine monthly vaginal examination to facilitate the detection and prevention of symptomless abortion and miscarriage.

Gonorrhœa as a complication of pregnancy is dealt with in seventeen lines. This might with advantage be considerably extended.

For the termination of pregnancy in pernicious vomiting the author gives pride of place to vaginal hysterotomy, and no mention is made of abdominal hysterotomy.

The description and diagrams of *trichomonas vaginitis* are exceptionally good.

The statement that uncomplicated retrodisplacements do not cause sterility or abortion will not pass unchallenged.

The chapter on accidental hæmorrhage leaves the impression that the severe varieties of the condition are the only ones. The comparative frequency and clinical features of the minor grades should be mentioned. The treatment of the severe grade recommended is immediate emptying of the uterus either vaginally or abdominally. The recognized conservative treatment directed to allaying shock and stimulating the uterus to contract should at least be mentioned.

The treatment recommended for *placenta prævia* will not meet with general approval. The author states that the uterus must be emptied with the least possible delay:

[1] "Practical Obstetrics for Students and Practitioners", by P. B. Bland, M.D., assisted by T. L. Montgomery, M.D.; 1932. Philadelphia: F. A. Davis Company. Crown 4to., pp. 750, with 516 illustrations, including 21 coloured plates.

Cæsarean section if the cervix is closed or nearly closed, or manual dilatation followed by instrumental delivery or breech extraction if the cervix is somewhat dilated. Bipolar podalic version is mentioned, but the two desiderata in dealing with this condition, namely, control of hæmorrhage and slow delivery, are not assessed at their true value.

The diagrams on the whole are good, and there are sufficient. Figures 152 and 153 show retained products being removed from the uterus without the use of a vaginal speculum. This might give an erroneous and dangerous impression. Figure 105 is a coloured plate purporting to be the appearance of the vaginal mucous membrane and cervix in *trichomonas vaginitis*, whereas it does not display the typical appearance at all clearly.

The publication as a whole does not provide anything that is not included in standard British text books, and it is doubtful whether this publication will be preferred in this country to them.

THE USE OF LIGHT IN TREATMENT.

"LIGHT THERAPY", by Dr. F. H. Krusen, traverses in a sane and rational manner the therapeutic application of all radiations from the infra-red to the ultra-violet regions of the spectrum.[1] The work shows a convincing array of accepted and proven facts, marshalled in an easily accessible form for reference, and supported by short *verbatim* quotations from the publications of other authorities. The case for these therapeutic agencies is well put and scientifically supported, with a pleasing absence of that unreasoning zeal which so often mars works on physical therapy.

Dr. Krusen is no intellectual slave who blindly assimilates current statements. It is usually assumed that ultra-violet light is not only useless, but even harmful to patients with pulmonary tuberculosis. He refuses to accept this view, and his conclusions thereon are reasonable, concise and energetic. No therapeutic method of any value is entirely free from potency to harm, and ultra-violet light is no exception to the rule. Whilst it is undoubted that evil may result from its injudicious use in pulmonary forms of tuberculosis, to condemn its application on such grounds alone is an obvious fallacy which confuses the relative significance of the therapy and the therapist. Skill and experience are just as necessary behind the lamp as behind the surgeon's knife.

The book is not over-burdened with useless pictures and descriptions of apparatus, but includes a brief and interesting summary of the more recent advances in instrumentation. There is a number of chapters headed "Systemic and Miscellaneous Diseases in which Ultra-Violet Light has been said to be of Value". The lists include such surprising items as appendiceal abscess, adenomata, hæmorrhoids, visceroptosis, pleural adhesions, *encephalitis lethargica* and osteoarthritis. These lists as a whole do not carry the stamp of the author's approval and would have been better omitted. The author's subsequent discussion gives just appraisal of all the legitimate uses of the radiation and thereby, perhaps, renders evident some latent quirk of deliberate humour in the "has been said" of the caption.

The final chapter summarizes the present position of light therapy under fourteen brief and pregnant headings. Considered as a whole, this small book is a well balanced and thoroughly reasonable summary of accepted knowledge and procedure in the field indicated by its title.

PHYSIOLOGY.

"STARLING" has for long been the greatest of British text books on human physiology, and under the editorship of Professor Lovatt Evans it admirably maintains the very high standard set by Professor Starling. In the preparation of the sixth edition there has been a thorough revision in all sections, and the book, as it stands, may be considered to present a thoroughly representative account of modern views in physiology.[2] It is not an easy book to read, and in the one thousand odd pages of the text an immense amount of material is presented. The average medical student would probably be lost in detail; it is essentially a book for an honours student. As a book of reference for a physician it would be invaluable.

A new feature of this edition is the insertion as footnotes and at the end of chapters of a few representative references for the guidance of students wishing to extend their reading.

RUSSELL HOWARD'S "SYSTEM OF SURGERY".

RUSSELL HOWARD'S well known text book of surgery, which now enters its fourth edition, still retains a conservative, essentially safe attitude towards surgery in general.[3] In that it is a reflection of the methods adopted by the clinical teachers of the London Hospital Medical School, it is of great value, for undergraduates especially. In addition, it is written clearly with that succinct expression which characterizes the best type of English teaching of clinical subjects. The author gives as accurate a correlation as possible between the symptomatology and the pathological picture. There are few waste words, few omissions of note, no overlapping, as is common in some systems, while the clinical definitions are short and clear cut. The main fault of the book is its extreme conservatism, although in this respect it must be admitted that only those methods known and tried by the writers have been given prominence. It is strange, however, to find no mention of the work of Kanavel on the fascial spaces of the hand, a contribution which has revolutionized the surgery of infections of the hand. The chapters on general subjects, particularly those on tuberculosis and syphilis, are full and good, but the nomenclature used for neoplasms is still rather archaic. The chapter on hernia—one of the best in the book—is practical and full, and contains much clinical lore, carefully and clearly expressed. It is, however, strange to find in the management of acute intestinal obstruction no references to the great importance of replacement therapy, as regards chlorides and fluids, nor to the remarkable results in the conservative treatment of intussusception in infants so well known in this country. Again, most surgeons, realizing the great importance of the grave effects of acute infections of the gall-bladder on liver function, which makes patients so affected such bad operative risks, will not agree with the advice to treat acute cholecystitis as an urgent surgical emergency, necessitating immediate operation. The omission, too, of the accepted Sauerbruch method of thoracoplasty as the most generally useful method of thoracic collapse, while other less valuable methods are given in detail, is also rather surprising. The chapter on neurosurgery is still very conservative, although the authors must have realized, with a progressive clinic of this type in their hospital, that this aspect of surgery has advanced far beyond the details given in this work. In spite of these defects there are so many good chapters that the book must retain its position as one of the best text books of clinical surgery in English, and it is one that can be safely recommended, particularly to undergraduates and general practitioners.

[1] "Light Therapy", by F. H. Krusen, M.D., foreword by J. A. Kolmer, M.D., Dr.P.H., S.Sc., LL.D.; 1933. New York: Paul B. Hoeber, Incorporated. Demy 8vo., pp. 300, with 33 illustrations. Price: $3.50.

[2] "Starling's Principles of Human Physiology"; Fifth Edition, edited and revised by C. Lovatt Evans, D.Sc., F.R.C.P., F.R.S.; the chapters on the Central Nervous System and Sense Organs revised by H. Hartridge, M.A., M.D., Sc.D., F.R.S.; 1930. London: J. and A. Churchill. Royal 8vo., pp. 1054, with illustrations. Price: 21s. net.

[3] "The Practice of Surgery", by R. Howard, C.B.E., M.S., F.R.C.S., and A. Perry, M.S., F.R.C.S.; Fourth Edition; 1933. London: Edward Arnold and Company. Royal 8vo., pp. 1345, with eight coloured plates and 584 illustrations in the text. Price: 30s. net.

The Medical Journal of Australia

SATURDAY, APRIL 28, 1934.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

STANDARDIZATION IN MEDICAL AND HOSPITAL PRACTICE.

During recent years it has been recognized that standardization is desirable in certain fields of human activity. The engineer and the industrialist know how necessary is standardization of their equipment and tools of trade. The average person possibly does not realize that standardization offers any advantages until, for example, he wishes to purchase an additional electrical fitting for his home and finds that the fitting he buys cannot be used with what he already has. Standardization may be looked on from the producer's point of view and also from that of the user. Obviously these points of view are closely related. If an agreement be reached that certain products of industry should be turned out according to a standard pattern, the producer needs less machinery than is necessary if he has to provide for patterns of many kinds; the time consumed in production is cut down; costs of production are less. In these circumstances the user, in addition to being able to stop worrying about multiplicity of patterns, is probably able to obtain a better article and has to pay less for it. Of course, there should be limits to standardization. An intelligent man would resent any ruthless attempt to make him live a life of unvarying routine, in which he was compelled to use none but standardized articles in a predetermined way. Such attempts—and in some countries attempts have been made—will not lead to either the contentment or the happiness of the individual. Special conditions of life—of work and of leisure—demand special treatment. It may be stated definitely, however, that in many directions standardization is necessary to the conservation of industry and is beneficial to the people of the community.

The Standards Association of Australia has done much useful work in many directions. This body was established in July, 1929, by the amalgamation of the Australian Commonwealth Engineering Standards Association and the Australian Commonwealth Association of Simplified Practice. It is under the ægis of the Commonwealth and State Governments; and amongst its objects is the following: "To prepare and promote the general adoption of standards in connexion with structures, materials, etc.; to coordinate the efforts of producers and users for the improvement of materials, processes and methods; and to procure the recognition of the Association in any foreign country." The Association has lately turned its attention to the preparation of standard specifications for materials and equipment for public institutions; particular attention has been paid to hospitals. Committees have been formed; and on these committees are official representatives of the British Medical Association, of hospital commissions and boards, of departmental and extra-departmental central purchasing authorities and of manufacturing organizations. Provision is also made for obtaining the assistance of independent experts. When a draft specification has been prepared by a committee of the Association, it is circulated widely for public comment, so that any person interested may express his opinion. Sometimes the opinions of manufacturing or administrative bodies

in Great Britain are obtained. In the light of all the comments received, a final review is made and the standard is promulgated. The work already undertaken by the Association regarding public institutions has to do with a range of textile goods, rubber goods, certain surgical appliances, hardware, furniture and so on.

There can be no doubt that the work of the Standards Association in regard to public institutions will be of immense benefit to hospitals and hospital practice. At the present time advantage must be taken of every possible means of keeping expenditure on hospital equipment as low as possible. This can be done without loss of efficiency and without detriment to industry and to those employed in it, if waste of effort is avoided. Effort is wasted when unnecessary variation in detail is sought. For example, sheeting and towelling, if manufactured on a large scale in fixed dimensions and of a prescribed quality, will cost much less than it will when every large hospital demands its own peculiar texture and its own particular width. When the Standards Association undertakes the setting up of a standard of surgical instruments, the standard will be set out on general lines. The Standards Association of Australia seeks for the sympathetic cooperation of medical practitioners; it will, we trust, not seek in vain. Many medical practitioners are members of hospital boards; many, though not occupying seats on hospital boards, have the ear of members of boards and of similar bodies; they can, if they will, use their influence to further work of great importance.

Current Comment.

THE CAUSE OF PEPTIC ULCER.

It would hardly seem that there is much more to be said about the ætiology of peptic ulcer in the present state of our knowledge, but, as Osler reminds us, it is good to take stock now and then, even if no fresh accretions of information have been made. Andrew B. Rivers has surveyed the subject with this end in view; he has not attempted to put up a new hypothesis or to bring fresh evidence in support of one or other of the current doctrines, but has tried to reduce the ætiological factors to a few simple categories that may be useful in managing the patient with the disease.[1]

The idea of Virchow and others, that a local circulatory disturbance is of importance in the genesis of ulcer, is more of interest in the consideration of the pathological mechanism than in the attempt to explain the onset of a clinical syndrome. It does, however, link up with the neurogenic factors, which are given prominence by Rivers in his discussion and in which our interest has been aroused keenly by such contributions as those of Cushing on the relation of peptic ulceration to lesions of the interbrain. This, then, is one of the chief categories referred to, that of the influence of the nervous system. The author points out that the industrial conditions prevailing in the world during the last few decades have been those of great stress. Of this America forms a conspicuous example, as he truly remarks, for there competition and keen business methods have called forth efforts prolonged and severe on the part of all who have carried high ambition and desire for success into their daily lives. Added to this has been the lamentable strain of the world depression, so that, if the neurogenic factor be an important one, opportunities have not been lacking to study it.

The other factors considered of importance are the capacity of hydrochloric acid to erode tissue, and that of trauma. As regards the former, Rivers points out that the baleful effect of acid will be manifest only while the concentration of acid is high and the defensive capacity of the tissues is low. When the balance swings in favour of the gastric or duodenal mucosa, healing will be favoured. This does not help us to an understanding of why the balance swings one way or the other, but the periodicity of ulcer, a phenomenon so well known to all who have to deal with the problem, is a fact beyond dispute. Trauma is a subject of many ramifications and includes the factor of infection, which, in acute ulcer at least, is often of known importance. Rivers aptly suggests that all the factors should be taken into consideration. Thus, as he shows, it is not uncommon for a syndrome to exist which simulates that present in peptic ulcer, but without any evidence of the presence of actual ulceration. In these cases he considers the local tissues immune, either from lack of accessory trauma or from superior resistance, even though the systemic stage is already set for the production of the ulcer picture.

The question of the so-called ulcer diathesis, emphasized by Draper, Hurst and others, he mentions, but is apparently inclined to lay more stress upon the general systemic factors as observed in each individual patient. In fact, in his general review he attaches considerable importance to this aspect of the problem, and in this will have the support of all those who try to treat the patient rather than the disease. The significance of any debilitating condition, such as chronic prostatic

[1] *Archives of Internal Medicine*, January, 1934.

disease, is also stressed. Such factors must be taken into account in assessing the causes of reactivation of ulcer, though whether they would be more likely to operate in virtue of their infective possibilities in connexion with trauma to the mucosa, or by a simple general lowering of bodily resistance, it is difficult to say.

Rivers puts forth a composite view; according to this, unfavourable systemic conditions predispose to a lowered resistance, infection and other trauma may diminish the local defensive powers of the mucosa, and finally a highly acid chyme adds the final and sustained push to the general attack. To this onslaught the tissues succumb; from it they rise healed, but not immune; and thus similar cycles may and do recur.

This is merely a clear statement of doctrines that surely would find few, if any, dissentients. But its value lies in its clarity and its simplicity. Every patient presents a problem like the hand at bridge; that "infinite monotony" of cards described by a Russian writer applies just as strictly to life and to clinical medicine, but with exactly the same degree of infinite variation within its infinite monotony. So each patient is an individual study. It is not enough to have fixed, even though elaborate, dietaries; it is not enough to follow an unvarying routine of Procrustean alkalis; it is not enough to examine and to remove the favourite and traditional septic foci, for it may be that the patient's private affairs, his business life, or even social or professional aspirations constitute an even greater bar to his recovery.

INTRACTABLE PAIN.

Amongst the difficult problems of medicine is that of pain. There is still much that is but little understood even in the explanation of varying degrees of pain in different persons, in its periodicity in the case of permanent and constant lesions, and even in the exact paths by which it is transmitted. There can be, however, no problem more clinically pressing in this field than the relief of intractable pain. Inoperable cancers, especially those with metastases, the severer types of neuralgia, and visceral disturbances of the anginal or tabetic categories may all cause such intolerable bouts of pain that the medical attendant is taxed to the utmost of his therapeutic resource. A number of suggestions have been made from time to time for the surgical relief of pain that is irremediable by less drastic measures.

Loyal Davis has recently reviewed this subject and has assembled in a compact article the neurological and surgical information available at the present time.[1] He points out that Ranson's work on cats has demonstrated that the fibres carrying painful stimuli are apparently non-myelinated and readily accessible in the posterior lateral sulcus of the spinal cord. This, however, refers only to the somatic fibres entering in the posterior roots, and inquiry has proved that visceral painful impulses can be blocked in the cord only by a complete transection. These impulses, passing up by the thoracic sympathetic trunk, travel by short relayed paths and avoid the lateral spino-thalamic tracts. Section of the right splanchnic nerve, however, successfully blocked them. Davis draws attention to the surgical bearing of these experimental facts, and remarks that the only successes gained in the relief of visceral pain by the operation of chordotomy have followed an incision including part of the grey matter, where the visceral pain relays are known to travel. Somatic pain, then, might be relieved by a limited cord section, but visceral pain, such as occurs in tabetic crises, needs a deeper section or cutting the posterior nerve roots. Linked up with the latter procedure is the knowledge that blocking of the cutaneous nerves is often effective in relieving internal pain. The mechanism involved in the function of the sympathetic in regard to pain conduction is doubtful, but the clinical success following section of the thoracic or abdominal trunks and the ablation of the cervical ganglia for different types of visceral pain is well known. Davis states that the relief of pain by section of sympathetic fibres is due to the interruption of efferent pathways. His paper, with its bibliography, should be consulted by those interested. But the general conclusions are simple and important. These are, to recapitulate, that chordotomy, the operation suggested by Spiller, will relieve pain of somatic origin only, unless the section is carried deeply enough to divide the subjacent grey matter; that section of the appropriate posterior roots will successfully relieve visceral pain, provided a sufficient number of roots be divided; and that the effect of operation on the sympathetic system is indirect, as indicated above.

Of course, it is only when faced by the problem of the relief of a constant and intractable anguish that operations of such severity as those designed to interrupt the sensory pathway would be mooted. But the reading of an account of the relief of symptoms in a case of *angina pectoris* of great severity by the performance of posterior root section leads us to hope that even patients so gravely affected may possibly obtain relief. Certainly the sufferers from incurable carcinoma should be considered in this light if it is impossible to relieve them in any other way. The indications may arise only at infrequent intervals, but when the customary analgesics are of no avail in cases of intractable suffering they should not be disregarded. In such instances it is of great importance to proceed upon right lines and to base any surgical treatment upon anatomical and physiological facts. It is one of the functions of the doctor to relieve pain, and occasionally in the exercise of this function he will find that desperate ills require desperate remedies.

[1] *The Journal of the American Medical Association*, December 16, 1933.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Respiratory Failure in Acute Poliomyelitis.

M. B. BRAHDY AND M. LENARSKY (*American Journal of Diseases of Children*, October, 1933) contribute their experiences and conclusions following the treatment of 46 children with respiratory paralysis due to poliomyelitis, in the Drinker respirator. They emphasize that all adjuvant medical treatment must be run to a time schedule and carried out as far as possible at the same hour. There must also be a fixed hour for turning the patient on his side and abdomen, though all future respirators will have a tilt-table to allow of postural drainage. Every effort must be made to avoid a rise of temperature within the respirator, even if it means running the air through an ice tank. The paralysed skeletal muscles need attention even in the respirator. It is necessary gradually to "wean" the child away from the machine by gradual lowering of the pressure with the motor still running and giving thereby some psychological comfort. The authors do not at present advise the treatment of patients with slight or moderate respiratory distress in the respirator, as damage to the elastic tissue of the lungs is always certain. They also advise an early return to bed as soon as sufficient spontaneous respiratory function has returned. Twelve of the patients with bulbar symptoms required the respirator; all died. A total of 46 children were so treated. Of those with spinal lesions only, twelve died in the respirator and from within six weeks of treatment. Two previous series have been reposted: that by Wilson, who had only three deaths among 23 patients, and that by Shaw, who had three deaths in a series of 10 patients with failure of all respiratory muscles; but in each case the criteria calling for the use of the respirator varied with the observer's views.

Thyreoid Disorders in Childhood.

RICHARD B. CATTELL (*The New England Journal of Medicine*, November 2, 1933) discusses the nature and relative frequency of thyreoid disorders in childhood from his own experience. He points out the rarity of endemic goitre in the United States of America. About 1% of patients attending his thyreoid clinic are under thirteen years of age. Developmental disorders are fairly frequent, including the lingual thyreoid—aberrant islets of thyreoid tissue in the lateral regions of the neck. Sporadic cretinism is rarely seen by this author, whereas colloid goitre is the commonest disorder of the thyreoid seen in children. He warns against the mimicry of true goitre by a prominent isthmus or layer of fat in young children. Careful preparation is the most important diagnostic procedure. He advocates conservative treatment by iodine and a well-balanced diet for these children. Primary hyperthyreoidism with or without goitre is not uncommon. The author has treated forty-five such cases. The clinical signs are identical with those of adults; but the basal metabolism test is unreliable in childhood. He advocates universal operation for these patients, and states that X ray therapy cannot be depended upon. The operation is done in two stages between which five drops or less of Lugol's iodine solution are given daily. He states that it is necessary to leave relatively larger remnants than are left in adult patients. Thyreoiditis is very rare in children. The only benign tumour of the thyreoid is the discrete adenoma, probably developing in fœtal rests.

Electrocardiographic Changes following the Subcutaneous Injection of Adrenaline.

M. M. MALINER AND W. E. MUNS (*Archives of Pediatrics*, October, 1933) investigated twenty children, aged five to fourteen years, with chronic valvular disease. Seven had initial insufficiency, five had initial insufficiency and initial stenosis, three had congenital heart defects, one aortic insufficiency, and four had possible heart disease. All children were tested two hours after the morning meal, in the recumbent position. An electrocardiogram was taken before and ten minutes after the administration of a 1 in 1,000 solution of adrenaline chloride in a dose of 0·06 cubic centimetre (one minim) per year of age. Five patients were used as controls. The patients to whom adrenaline was given had an average increase in pulse rate of 7·9 per minute; in five cases the pulse rate became slower, on an average 2·7 beats per minute. In the control group there was an increase of two to six beats per minute. One patient developed sinus arrhythmia, another premature contractions. Electrocardiographic changes were mainly in the degree of voltage of the ventricular complexes. The *P* deflections showed a higher voltage in 20% of the cases and lower in 40% of the cases; the *R* deflections showed a higher voltage in 33% and a lower in 53%; the *T* deflections a higher voltage in 13% and lower in 66%. In one-third of the cases there was a decreased *P R* interval, and in 13% a shortening of the *Q R S* interval. Even in two of the control cases there were decreased voltages, so that either the adrenaline had no effect on the voltages or perhaps a lower voltage at times accompanies an increased tonus of the heart muscle. No disturbances were noted in the *R T* or *S T* intervals. The clinical effects of adrenaline injection previously noted in these patients, namely, tachycardia, arrhythmia, appearance of latent murmurs *et cetera*, have little or no electrocardiographic counterpart.

Rickets and Hyperparathyreoidism.

BENGT HAMILTON AND CHARLES SCHWARTZ (*American Journal of Diseases of Children*, October, 1933) emphasize the fact that the disturbance of calcium and phosphorus metabolism in rickets is only partially reflected in the concentration of these substances in the serum. While a low phosphorus concentration is one of the most constant findings in the active stage of the disease, the concentration of calcium is generally normal or only slightly decreased, though this calcium level is rather precariously maintained. The giving to rhachitic animals of a solution of an easily soluble calcium salt by mouth and the resulting increase in the serum calcium content is often marked enough to cause the death of the animal from hypercalcæmia. Hypertrophy of the parathyreoid glands occurs in rickets; the authors have attempted to establish the existence of an increase in parathyreoid hormone in the plasma of rhachitic animals, in this case rabbits. They conclude from their observations that the blood of rhachitic rabbits is rich either in parathyreoid hormone or some other substance with identical effect on the serum calcium, and also that the marked affinity for calcium in rhachitic blood is due to hyperactivity of the parathyreoid glands.

Rickets in Puerto Rico.

MARTHA M. ELIOT AND EDITH B. JACKSON (*American Journal of Diseases of Children*, December, 1933) have studied 584 Puerto Rican children from one to thirty-four months of age in order to observe the Röntgenographic appearance of the bones of infants living under the influence of tropical sunlight, and to make a comparison with similar findings in infants living in a temperate climate, namely, New Haven, Connecticut. Rickets is undoubtedly a rare disease in Puerto Rico. By Röntgenographic means only five cases of rickets (1% of the total) were detected. There is a similar absence of slight deviations from normal, termed mild rickets, which occurs in temperate climates. The rate of growth in Puerto Rico is slower than in the United States of America, and this may affect the incidence of the slighter manifestations. Doubt is thrown on the reliability of the clinical diagnosis of rickets in the absence of Röntgenographic evidence of the disease. The average calcium content of 32 samples of blood examined was 10·6 milligrammes per 100 cubic centimetres of serum; the average inorganic phos-

phorus content of 34 samples was 5·2 milligrammes. Of the children examined, 10% had osteoporosis that was associated with malnutrition and stunted growth. In one-third of the skiagrams of all children transverse lines in the radius and ulna were noted.

ORTHOPÆDIC SURGERY.

Treatment of Fractures and Fracture Dislocations of the Spine.

R. WATSON JONES (*The Journal of Bone and Joint Surgery*, January, 1934) describes the results of treatment by his method in eighty cases of crush fracture of the spine. His method, which requires no special apparatus, skilled assistance, manipulation of the spine, or anæsthetic, is ambulatory throughout. The risk of lung complication and functional disorder is minimized, and the method is equally applicable to the difficult high dorsal fractures and cervical fractures. Comminuted fractures are easily reduced. They consolidate slowly and require six months' immobilization. The author considers that fractures with paraplegia demand immediate reduction without anæsthesia. His routine has replaced laminectomy for spinal fracture. Recovery is secured in 70% of low dorsal and lumbar fractures, but is rarely seen in high dorsal fractures. The only contra-indication to hyperextension treatment is the very rare comminuted hyperextension fracture of the vertebral body. For first-aid treatment he considers that the patient must be transported in a prone position. Many fractures have undergone spontaneous reduction before being seen by the surgeon. After reduction they may be radiographically indistinguishable from normal. Herein, he states, lies the explanation of Kümmel's disease.

Visceral Lesions in Proven Bone Tuberculosis.

C. H. SYNDER (*The Journal of Bone and Joint Surgery*, October, 1933) calls attention to the fact that bone and joint tuberculosis is a systemic disease. He stresses the point that a positive diagnosis is essential to correct treatment and accurate interpretation of results. Of 164 consecutive cases seen during the past year clinically diagnosed as bone and joint tuberculosis, 100 were established cases, 50 had no pathological or bacteriological reports, and in 14 the guinea-pig inoculation and pathological tests were productive of no reaction. Pulmonary lesions in substantiated bone and joint tuberculosis average 40% in the author's series, 27% with adult pulmonary or parenchymal lesions, and 13% with active pulmonary tuberculosis of childhood. Eight *per centum* of his patients had renal tuberculosis. He urges more frequent urinalysis, guinea-pig inoculation and special search for tubercle bacilli, in the investigation of patients with an acid urine containing no organisms, but in which white and red blood cells are found by the usual staining methods. He considers that failure of the skin to react to the tuberculin test, especially to repeated increasing dosage of a recently prepared or fresh solution of old tuberculin, is of definite diagnostic value.

Old Dislocations of the Shoulder.

W. R. CUBBINS, J. J. CALLAHAN AND C. S. SCUDERI (*Surgery, Gynecology and Obstetrics*, February, 1934) describe their modification of Nicola's operation for the treatment of old dislocations of the shoulder joint. The patient is placed in a semi-sitting position on the table and an incision is made between the anterior edge of the deltoid and the upper border of the *pectoralis major*, extending up to the middle of the clavicle, then around the acromion and the lateral five centimetres (two inches) of the spine of the scapula. The skin flap and deltoid muscle are reflected down, thus giving a free exposure of the displaced head and the flattened capsule. Subsequent steps of the operation depend on the amount of thickening of the capsule and consequent obliteration of the cavity of the joint. The authors reflect the greater tuberosity in order to get free exposure of the joint, and, having divided the tendon of the long head of the biceps muscle, place it through a hole drilled in the head of the humerus and then up through a smaller hole through the acromion process, where it is fixed; this anchors the head. The deltoid is accurately re attached to its origin and the skin incision closed, the arm being held in the position of salute in a cast from four to six weeks.

Recurrent Dislocation of the Jaw.

LEO MAYER (*The Journal of Bone and Joint Surgery*, October, 1933) states that intractable slipping at the temporo-mandibular articulation may be due either to a lesion of the inter-articular fibro-cartilage corresponding to a lesion of the internal meniscus of the knee, or to an actual dislocation of the condyle, which slips forward over the *eminentia articularis*. The former group can be cured by the simple removal of the meniscus. For curing the latter group a new operation is described, consisting in the construction of a bone block just anterior to the *eminentia articularis*. Four instances of cure are cited, one of the first group and three of the second.

Block Osteotomy of the Femur.

E. W. RYERSON (*Journal of Bone and Joint Surgery*, October, 1933) states that abnormal curvature of the femur, due to fracture, rickets, or other causes, usually requires operation for its correction. A transverse osteotomy, while easy to perform, is decidedly not easy in its after-care. The maintenance of proper alignment and apposition requires constant attention and considerable surgical ability. Cuneiform osteotomy, the removal of a wedge of bone, affords greater stability, but causes a distinct loss of length. He considers a curved osteotomy is mechanically preferable, but is difficult to perform with accuracy.

Fracture of the Neck of the Femur.

G. W. LEADBETTER (*Journal of Bone and Joint Surgery*, October, 1933) believes that whatever method of reduction and fixation is selected, the so-called after-care is the most important in the preservation of life of persons affected with fractured neck of the femur. He recommends plaster fixation, properly applied, as the best means of carrying out treatment. Plaster fixation must have two objectives: first, to immobilize the fracture, and secondly, to facilitate post-operative care. The plaster should be applied tightly, and the hip reduced and fixed in the proper degree of abduction and internal rotation; a layer of glazed cotton is placed about the trunk and thigh from the level of the nipples to a point about half way between the hip and the knee. The author discredits treatment by the various traction methods or by the so-called sandbag method, which he considers no method at all. The injured leg is flexed at the hip at 90°, with the leg at an angle of 90° to the thigh. Direct manual traction in the axis of the flexed thigh is then made, together with slight adduction of the femoral shaft. In this position the thigh is internally rotated approximately 45°. The leg is slowly circumducted into abduction, the internally rotated position being maintained. The amount of abduction varies with the individual and can be measured accurately, representing the difference in degrees of the angle made by the fractured neck with the shaft and the angle between the neck and the shaft on the normal side. The test which in his experience has indicated that the fracture has been completely reduced is as follows. After the leg has been brought down in the measured degree of abduction and internal rotation, the heel of the injured leg is allowed to rest on the outstretched palm. If the reduction is complete, the leg will not evert itself. Should there be no interlocking of the fragments, however, the leg will slowly rotate externally. The author considers that his method of manipulation is anatomically and physiologically logical and easy to accomplish, and, if the test which he believes to be infallible is applied, assures good anatomical reduction, thereby greatly enhancing the chances of bony union by preserving the normal relations of the only source of callus in the neck of the femur, namely, the cancellous structure. He considers that when medical practitioners free themselves from didactic and empirical teachings and arrive at agreement on better methods of complete fixation of these fractures, the percentage of good end-results will increase.

British Medical Association News.

SCIENTIFIC.

A meeting of the Queensland Branch of the British Medical Association was held at the Hospital for Sick Children, Brisbane, on March 2, 1934. The meeting took the form of a series of demonstrations by members of the honorary staff.

Cretinism.

Dr. S. F. McDonald showed two patients suffering from cretinism, to illustrate the poor response to inadequate dosage of thyreoid extract in treatment. The first was a child, aged ten months, who showed all the signs typical of cretinism. The patient was having 0·03 gramme (half a grain) of thyreoid extract daily, which was not sufficient. The amount was to be increased. The outlook was fair.

The second patient was a woman, aged twenty-five years, who had had no treatment till a few months previously. Her mental age was that of a child of about four years. She had the condition of the skin and hair and the anæmia typical of the condition.

Microcephalic Idiocy.

Dr. McDonald's third patient was a child, aged seven years, who was a microcephalic idiot. The child was utterly helpless, made no attempt to talk, and took no interest in anything; he would not take solid food and was unclean in his habits. There were two other children in the family who were also idiots.

Mongolism.

Dr. McDonald also showed a mongol, aged nine months, who was the last child of a large family. This child was very small and his appearance was typical of mongolism. He had suffered from digestive trouble with stenosis at the ileo-cæcal valve, and had a distended stomach and the usual ladder-pattern intestine. Thyreoid extract was of no use whatever in this type of case.

Common Skin Conditions.

Dr. W. Heaslop showed patients suffering from various common skin conditions. A girl was shown who had suffered from ringworm of the head. The hair had been straight and scanty. X rays were applied to the scalp to cure the ringworm and the hair was now a curly mass.

The next patients were two boys with typical ringworm of the body.

The last patient was a boy suffering from *impetigo contagiosa*. Dr. Heaslop remarked that this condition was seen among school children, particularly at the beginning of summer. The stages were pimple, vesicle, pustule and crust; the primary cause was a streptococcus which lived for a few hours only; invasion then took place by staphylococci. The treatment consisted in bathing with perchloride of mercury solution (one in ten thousand) or weak boracic lotion, and then applying 1% ammoniated mercury ointment.

Umbilical Hernia in Children.

Dr. K. B. Fraser stated that there were two varieties of umbilical hernia: (i) congenital and (ii) post-natal, developing subsequently to the ligature of the cord at birth. The first variety of congenital hernia, known as exomphalos, was a very rare condition due to a developmental error. The sac was large, thin-walled and cone-shaped, and contained small intestine always, and sometimes stomach and liver. Immediate operation offered the only hope of cure. Post-natal hernia, on the other hand, was a common condition and one in which the importance of adequate treatment seemed hardly to be realized. The hernia generally appeared soon after birth, and was due either to persistence of a small protrusion of the umbilical cœlom or imperfect closure of the embryonic *linea alba* just above the umbilicus. The only condition it could be mistaken for was an epigastric hernia, which was a pedunculated fatty tumour with no fibrous ring, situated in the mid-line above the umbilicus. The sac was usually oval in shape, with a comparatively narrow neck, which could be felt passing through a firm fascial ring; this ring was generally circular, but sometimes was elongated horizontally and narrowed vertically.

The correct rationale in treatment was of great importance; in this respect the size of the ring was a guide rather than the size of the hernial sac. In young infants the umbilicus should be examined repeatedly; the hernia would then be discovered immediately on its appearance and appropriately treated. There were three recognized methods of treatment which were used at the Hospital for Sick Children: (i) mechanical obstruction of the ring (mechanical occlusion), (ii) conservative operation, (iii) radical operation. If the hernia was discovered soon after birth, mechanical occlusion gave good chances of success, even if the ring was rather large, and should always be tried. The earlier adequate treatment was instituted in any given case, the better the chances of recovery. There were different methods of mechanical occlusion. Inversion of the abdominal wall with a small strip of adhesive strapping had some value as a prophylactic agent, but had very little value once a definite hernia was established. Rubber trusses had the disadvantage that it was almost impossible to keep the button directly over the hernia all the time. Pennies and cardboard pads were too flat and were prevented by the surrounding fatty tissue from fitting accurately over the aponeurotic ring. The patent tops of soda-water bottles were better, but they had the disadvantage of being a fixed size. The best appliance was a piece of circular cork from four to eight millimetres in depth, with a diameter small enough to fit well down on the neck of the sac, and large enough completely to overlap the firm fibrous edge of the fascial ring. This diameter varied from twelve to twenty-two millimetres. The cork was applied carefully after the hernia had been reduced, and was held in place by ordinary adhesive strapping, which completely encircled the body. The baby could be bathed as usual; the strapping was examined at least once a week and changed as soon as it loosened or if there were any sign of skin irritation; it would generally stay on for three weeks. The edge of the cork next the skin should be slightly bevelled, in order to prevent skin abrasion. The cork should never be made cone-shaped, as the apex of the cone fitted down into the ring and tended to widen it.

Mechanical occlusion, when decided upon, should always be persevered with for three months. In patients over six months of age there was not so much hope of success with this method; but it should always be tried on patients up to the age of fifteen months, provided the ring was small. Over the age of fifteen months there was little hope of a real cure by mechanical means alone.

If this method failed, the conservative operation was worthy of trial if the ring would not admit the tip of the little finger, or if the ring were not more than approximately eight millimetres in diameter. This operation consisted in subcutaneous ligature of the neck of the sac and involved little risk if done carefully. If this method were not feasible or had been tried without success, recourse must be had to radical operation. This was not a particularly easy operation in children and should never be done on a child under two years of age, owing to the possibility of massive lung collapse. The results, however, fully justified its use in cases which failed to respond to less radical methods.

Dr. Fraser then showed several children who had been treated by the different methods.

Dr. L. G. Hill then demonstrated the method of applying strapping with a cork over an umbilical hernia. He said that the success of the method depended on correct application; several points to remember were: the width of the strapping, the cork and its size relative to the hernia, nicks in the strapping, and the length and direction of the strapping.

DR. J. G. AVERY said that the operative treatment of umbilical hernia was either conservative or radical. The conservative operation should be done only if the size of the ring in the aponeurosis were less than 0·8 centimetre (one-third of an inch) across, and if the surgeon were sure that the contents of the sac were completely reducible. The apex of the sac was caught in tissue forceps and held up while three radiating equidistant incisions were made down to the aponeurosis at the neck of the sac. A tunnel was then made with a dissector from one incision to the next all round, keeping as close to the sac and to the aponeurosis as possible. A thick catgut ligature was then threaded round the neck of the sac in the tunnel and tied. The skin wounds were closed and a dressing applied. Adhesions formed in the sac and the closure of it tended to hasten the natural closure of the aponeurotic gap.

The radical cure should be done if this operation failed, or in children over three years of age, or if the sac contents were irreducible. One method was to divide umbilicus and sac by a transverse incision, down to the aponeurosis, and to dissect out a cuff of peritoneum down to the neck and tie it off. The aponeurosis was then cleared and defined on either side, a small lateral incision was made into each side of it, and flaps were defined and sutured to each other in an overlapping fashion.

Another method was to make a curved incision extending round above the umbilicus and to dissect carefully down to the sac, which was opened and cut across; the neck, having been cleared, was ligatured with catgut. The aponeurosis was treated as already mentioned, and the flap was sutured back in position. By this method the umbilicus was preserved in its entirety.

After-treatment of the radical cure consisted in rest in bed for three weeks and the wearing of a strapping or rubber binder for some time afterwards.

Dr. Avery then showed a boy, aged nine months, who had been admitted to hospital on November 22, 1931. He was wasted and had to be fattened for three weeks prior to operation, which was done on December 12, 1931. The hernia had been strapped for some time previously, but was elongated and prominent, being 1·9 centimetres (three-quarters of an inch) wide and 3·1 centimetres (one and a quarter inches) long. At operation a lateral incision was made on each side of the umbilicus and was deepened to the sheath of the rectus. A catgut ligature was inserted round the neck of the sac and tied.

Marasmus.

DR. SHIRLEY LANE showed a girl, aged eight months, whose birth weight was unknown, but who had had "gastritis" at the age of four months. She had been fed on "Lactogen", given every two hours. She had been admitted to hospital on December 16, 1933, when her weight was 3·7 kilograms (eight pounds four ounces). She was given milk and water in the proportions of one and three, taking 120 cubic centimetres (eight fluid ounces) at a feed, and having seven feeds in twenty-four hours. The strength of the mixture was gradually increased until she was having equal parts of milk and water with four grammes (one drachm) of sugar to 30 cubic centimetres (one fluid ounce) of mixture. On December 18, 1933, blood examination revealed 2,710,000 red blood cells per cubic millimetre and a low hæmoglobin content. On January 13, 1934, an injection of "Hepasol" was given. Six of these injections in all were given up to February 2, 1934. The red blood cell count had risen to 3,450,000 per cubic millimetre; but the hæmoglobin value had remained the same. The weight had increased to 5·5 kilograms (twelve pounds four ounces).

Dr. Lane's second patient was a girl, aged five months, whose birth weight had been 2·9 kilograms (six pounds eight ounces) and who on admission to hospital weighed 4·3 kilograms (nine pounds nine ounces). She had not gained weight on various feedings and was eventually given one part of milk with three parts of water and two grammes (thirty grains) of sugar, having seven feeds of 180 cubic centimetres (six fluid ounces) each in the twenty-four hours. The quantity of food was increased by 30 cubic centimetres (one fluid ounce) of milk and four grammes (one drachm) of sugar each day, and in three days feeds of 240 cubic centimetres (eight fluid ounces) were taken well. There was steady gain in weight of 11·0 grammes (four ounces) per week, and the child was discharged in two weeks.

Dr. Lane said that the condition of marasmus was characterized by stationary weight or actual loss of weight, so that at four, six or eight months the child might weigh little more than at birth. There might or might not be vomiting and diarrhœa or "hunger" stools. Marasmus more often affected bottle-fed babies and was chiefly due to a long period of under-feeding or wrong feeding. In appearance the child was wasted, pale and atonic, and had a relatively very large abdomen with loss of elasticity of the skin. Intercurrent affections were common and often fatal. These cases had in common a history of bottle feeding; the patients were all markedly under-weight and anæmic, and they lacked signs of any other abnormal condition. One, as often happened, had been tried on many kinds of food without improvement, and here Dr. Lane thought it would be agreed that it was usually better for the physician to pin his faith on one food, such as cow's milk, and try to get various modifications to suit the infant, rather than to swing from one food to another. The diet given to these infants on admission had been one part of milk to two parts of water, with the addition of sugar in increasing quantities. As soon as the milk and water were well tolerated, egg yolk, broth and orange juice were added.

Malnutrition in Older Children.

DR. PHYLLIS CILENTO showed a boy whose history began in infancy with whooping cough at the age of eight months. The mother stated that he had been delicate always. At the age of three years he had attended the out-patient department of the Hospital for Sick Children because of obstructed nasal passages, snoring and constant colds. At six years he was admitted with faucial diphtheria following a week's illness. At ten years he had had an attack of vomiting and diarrhœa, and at ten and a half years, measles and influenza. When aged eleven years he had complained that his legs were so weak in the morning and that he could not stand on them for any length of time. The hundred yards walk to school tired him and he could not drill for the allotted ten minutes. Physical examination revealed no abnormality and advice was given regarding diet and exercise. Most of his ills were considered imaginary. He still suffered from nasal obstruction and constant colds; enlarged tonsils were removed in August, 1933. His present symptoms were that, though improving, he still had "weak faint turns" with occasional attacks of vomiting and pains in the head. He was always tired and languid and had a very poor appetite; he suffered from frequent coughs and recently a puffiness round the left eye. His feeding history began before birth. The mother was very poor while she was pregnant and lived mainly on bread and dripping, having no milk. There was little breast milk for the sickly baby, who weighed 2·9 kilograms (six and a half pounds) and he was soon weaned and given "Lactogen" and condensed milk. Apparently he had never had an adequate diet, having very little milk and refusing vegetables. At present his staple food was white bread and jam or syrup, a little meat, sometimes butter. As thirty-three shillings and ninepence had to be shared between parents and three children, and the weekly rental was twelve shillings and sixpence, little was left for food. One pint of milk a day was used between them, and powdered skimmed milk for the rest.

The boy, aged eleven and a half years, was pale, thin, and of poor muscular tone and development. His weight six months before the meeting was 24·3 kilograms (three stone twelve pounds), the normal for his age being 35·1 kilograms (five stone eight pounds). His height was 135·3 centimetres (four feet five and a half inches), a little under normal. His chest measurement was 55·0 centimetres (22 inches), the equivalent of a child of four years, and he had a chest expansion of 1·25 centimetres

(half an inch). No abnormality was detected on examination of his heart and lungs, though at one period the apex beat had been 1·25 centimetres (half an inch) outside the nipple line. The knee jerks were present, but sluggish. There was no reaction to the Wassermann and Kahn tests. The urine was acid in reaction and the specific gravity was 1·028; albumin and sugar were present. The child's condition improved greatly with treatment by cod liver oil, *syrupus ferri phosphatis compositus*, and liquid extract of malt. Exercises were ordered. The weight increased to 25·2 kilograms (four stone) in six months. Dr. Cilento remarked that it had been impossible to prescribe an adequate diet—the main indication for treatment—owing to the economic circumstances and the mother's limitations.

Dr. Cilento said that this child approached the incipient beri-beri type. Although there was no definite deficiency disease, he was in that "twilight zone" (McCollom) of nutritional instability and would be only a "C_2" citizen.

Dr. Cilento's second patient was a child, aged four years, who illustrated a further stage of the effects of undernourishment in infancy. This girl had now reached early childhood and was 3·0 to 3·6 kilograms (seven to eight pounds) under-weight, although she lived in the country where plenty of milk and fresh food were available. The child was a premature baby and was always small, shy and nervous; she cried and vomited with nervous excitement. The appetite was always poor; she was always tired and could not run about or play with other children without extreme fatigue. She had frequent colds and sores on the face and neck; she suffered from discharging ears when teething, and from pyelitis. She was a restless sleeper and suffered from night sweats and night terrors, and showed nervous instability and a tendency to infections.

The mother had vomited all through pregnancy and had had no milk or vegetables. She fed the baby at the breast for fifteen months; but her diet consisted only of meat, white bread, jam, sweet potatoes and cabbage, usually cooked with soda. The child never drank milk or ate vegetables, except potatoes and pumpkin; she ate meat, including sausages, but very little fruit. The diet was deficient in calcium and the vitamins *A*, *C* and *D*. Marked improvement resulted from the administration of a milk diet, cod liver oil, the syrup of calcium lactophosphate, and potassium citrate.

Congenital Dislocation of the Hip.

Dr. L. W. Gibson showed two children suffering from congenital dislocation of the hip. The first, aged eleven years, had double congenital dislocation; the second, aged seven years, had congenital dislocation of the right hip.

Dr. Gibson said that congenital dislocation of the hip was one of the many orthopædic conditions in which early diagnosis was of paramount importance from the point of view of successful treatment. The condition was rarely recognized before the child began to walk, and one of the first things that should arouse suspicion was that the child was late in walking. If there were any doubt about the cause of delay in walking, an X ray examination should be made. The child with double dislocation had a waddling gait, the pelvis lurching sideways and forward at each step. The other child had a definite limp, with a lurching of the body to the affected side. The limp was due to shortening, which might be as much as 2·5 or 2·75 centimetres (one or one and a half inches), and the lurching was due to the instability of the joint as a weight-bearing member and to the loss of abduction. The Trendelenburg sign was easily illustrated in these cases; if a normal child stood on one leg and flexed the thigh on the abdomen, the pelvis was raised on the side on which the knee was flexed, thus allowing the centre of gravity to be moved over the weight-bearing axis. If the hip of the standing leg were dislocated, the pelvis dropped on the opposite side. This was because the power of abduction was insufficient to tilt the pelvis upwards, and it hung on the upper end of the femur, suspended, as it were, by its muscle attachments. The peculiarity of gait was due to a repetition of this procedure at each step. Shortening was present, and it was usually found that the trochanter was displaced upwards and backwards and about 2·5 to 3·75 centimetres (one to one and a half inches) above Nélaton's line. The head of the femur could sometimes be palpated on the outer and upper aspect of the ilium, and there was a depression in front of the joint. Movement was limited somewhat in abduction, but other movements were abnormally free, and telescoping of the thigh could often be elicited, due to the ease with which the femur could be displaced upwards or downwards on the side of the ilium. This was often less apparent in the older children on account of secondary contractures of the soft tissues. In double dislocation there was always a marked lordosis, due to the posterior displacement of the axis of suspension of the pelvis, and there could usually be seen a widening of the perineum with a prominence of the trochanters on the lateral aspect of the buttocks. An X ray examination clinched the diagnosis.

Dr. Gibson remarked that an error in diagnosis of this condition was generally one of omission and not commission. In the differential diagnosis there had to be considered: fracture of the neck of the femur or destructive disease of that part, when the trochanter would be elevated above Nélaton's line; also a traumatic *coxa vara* due to slipping of the epiphysis. A positive Trendelenburg sign might be given by three conditions besides the congenital deformity: (i) ankylosis of the hip in abduction; (ii) poliomyelitis involving the lateral abdominal muscles or abductors of the thigh, when the inability to tilt the pelvis upwards was due to muscle weakness; (iii) a severe *coxa vara*, either rhachitic or traumatic, causing marked limitation of abduction by the impinging of the great trochanter on the ilium.

The waddling gait due to double congenital dislocation was characteristic. In the gait seen in pseudo-hypertrophic muscular dystrophy the patient tended to lean backwards from the hips in an attempt to regain balance. Spondylolisthesis was characterized by pronounced lordosis, but it was rare in children, and the hip joints were normal.

Dr. Gibson stressed the desirability of an X ray examination when there was a possibility that delay in walking was due to a congenital hip deformity or when a persistent limp occurred after the child had begun to walk.

Intraocular Hæmorrhage Following a Blow on the Eyeball.

Dr. E. O. Marks showed a boy, aged eight years, who had been bumped by his small brother's head on the left eye, early on the morning of February 13, 1934. The sight of the right eye had been lost as a result of a perforating injury from a stone in the previous April. During the day of February 13 little inconvenience was apparently felt, the boy playing as usual. During the night, however, he complained of pain and sickness. There was blood in the anterior chamber and the sight was reduced to a mere perception of light. The following morning he was taken as soon as possible to Bundaberg, a distance of forty miles. A medical practitioner there sent him to Brisbane by the earliest train. At the time of his admission to the Hospital for Sick Children on February 15 the anterior chamber was full of blood and the intraocular tension was markedly increased. A large quantity of blood was evacuated through a keratome incision at the upper margin of the cornea. For two days fresh blood continued to appear in the anterior chamber and to ooze from the incision. This ceased after the administration of hæmostatic serum. The boy was also given thyreoid extract, at Dr. J. Lockhart Gibson's suggestion. Hypertension did not recur as the wound healed; but the iris and pupil remained obscured as if the anterior chamber were filled with dark blood. Not until February 26, eleven days after the paracentesis, did the anterior chamber show signs of clearing, the iris becoming visible through the periphery of the cornea, which elsewhere still had a deposit of blood on its posterior surface. Hand movements could now be distinguished.

There was some possibility of a hæmophilic tendency, the father stating that the boy was always very liable to bleed from cuts *et cetera*. Prior to the recent injury to the left eye, the father had had some suspicion that the sight was not as good as formerly, and was intending to have the eye examined. Dr. Marks said that one could only speculate whether this possible visual defect had any bearing on the present disaster. The case illustrated a not uncommon problem, which always caused anxiety as to the best procedure to adopt. While small hæmorrhages in the anterior chamber usually became absorbed with little permanent harm, and extensive hæmorrhages with marked hypertension obviously demanded paracentesis, there were many cases between these two extremes of extensive hæmorrhage without marked hypertension. Some cleared up and some left a permanent brown staining and opacity of the cornea, with a useless eye. Dr. Marks asked when paracentesis should be done, with its risk of fresh hæmorrhage, and when the surgeon should hold his hand.

Foreign Bodies in the Ear and Nose.

DR. KENNETH GREEN demonstrated the removal of foreign bodies from the nose and ear. He said that this removal, in a child, often presented a problem, sometimes difficult, even with all facilities at hand. There were two main types of foreign body: (i) those which swelled by contact with secretions, such as seeds, pieces of corn *et cetera*; (ii) those which did not swell, such as beads, buttons, pebbles *et cetera*. There was sometimes a history that the child had put something into its nose or ear; sometimes the foreign body was found when the child was being examined for obstruction or discharge from the nose, or for earache or aural discharge. A unilateral discharge from the nose, especially if blood-stained, was usually due to a foreign body. Frequently the discharge in such cases was found to contain the Klebs-Löffler bacillus. Once the foreign body was removed, the discharge ceased, and further examination revealed no diphtheria bacillus. Nasal diphtheria alone most frequently produced a bilateral discharge and constitutional symptoms. The medical practitioner should always believe a child that said it had put something into its nose or ear, and he should not believe it if it denied having done so until examination had satisfied him that there was no possibility of a foreign body being present.

Dr. Green went on to say that the first thing to be done if the presence of a foreign body in the ear was suspected was to inspect the ear with a good light. Cerumen might obscure even a recent foreign body. The position of the foreign body being seen, it was wise to drop in some absolute alcohol; this could do no harm and would at least help to sterilize the canal. If the foreign body were of the first type mentioned above, it was essential not to make any attempt to remove it until it had become shrunken by the action of alcohol. Removal could nearly always be effected by syringing, though this might have to be repeated more than once. It was essential to use a metal syringe, capable of holding 120 cubic centimetres (four fluid ounces), and having finger and thumb rings, so that considerable force could be given to the stream of water. A general anæsthetic would often be necessary to allow of thorough syringing. If repeated syringing failed, the foreign body was usually deep to the isthmus, and its extraction might be extremely difficult. Hoot's or crocodile forceps might be useful for passing into the hole of a bead, or grasping some projecting part of a foreign body; but the greatest care must be taken not to lacerate the walls of the meatus. It was worse than useless to attempt to grasp any smooth, round body with such instruments. Retained foreign bodies, if syringing was repeated, did not lead to the complications that ill-advised attempts at removal with instruments did. It might be necessary to reflect the auricle and cartilaginous meatus forward and to remove some of the posterior bony meatal wall, to get at a deeply situated foreign body.

With regard to foreign bodies in the nose in children, Dr. Green remarked that a general anæsthetic might be necessary for a satisfactory examination. If the foreign body could be seen, there was one simple manœuvre permissible without a general anæsthetic, and this was usually successful. It was to pass a probe, flat end first, beneath the foreign body and well back beyond it. The free end of the probe was then pressed downwards and the foreign body was usually levered out of the anterior naris before the child realized what was happening. If this failed and the foreign body was firmly impacted and inaccessible, a light general anæsthetic was given, the nasal mucosa shrunken with adrenaline and the foreign body gently removed with a hook or crocodile forceps; if too large, it might have to be broken with strong forceps and removed piecemeal. Every effort should be made to avoid increasing the damage already done to the nasal mucosa and turbinals by the foreign body, as adhesions readily formed and might seriously obstruct the nasal passage later.

Post-Graduate Work.

LECTURES IN MELBOURNE.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE announces that arrangements have been made with Professor David Barr, Professor of Medicine, Washington University, St. Louis, United States of America, to deliver a series of post-graduate lectures in Melbourne in July and August next.

It will be remembered that Professor Barr's colleague, Professor Evarts Graham, delivered a highly interesting series of lectures on certain aspects of gall-bladder and lung surgery under the auspices of this committee in 1930. It is due to his cooperation and interest that the Committee has been able to make arrangements with Professor Barr.

Professor Barr is widely known as a physician, particularly in regard to his work on endocrinology, on which he is a recognized authority. He is the author of the chapter on diseases of the parathyreoids in the "Oxford Medicine". In addition, his personal observations during the epidemic of encephalitis in St. Louis in 1933 were extensive and will form the basis of one lecture.

The time-table is as follows:

Monday, July 23, 1934: "The Functions of the Anterior Lobe of the Hypophysis."

Wednesday, July 25, 1934: "The Suprarenal Glands and Addison's Disease."

Friday, July 27, 1934: "Thyreotoxicosis."

Monday, July 30, 1934: "Hyperinsulinism and Related Conditions."

Wednesday, August 1, 1934: "The Relation of the Parathyreoid Glands to Calcium Metabolism."

Friday, August 3, 1934: "Epidemic Encephalitis."

The lectures will be delivered in the Medical Society Hall, Albert Street, East Melbourne, at 8.30 p.m. Further details will be announced subsequently. The Honorary Secretary, 61, Collins Street, Melbourne, will be glad to answer any inquiries.

ANNUAL REFRESHER COURSE IN MELBOURNE.

THE annual refresher course will be held at Melbourne during the fortnight Monday, July 23, to Friday, August 3, inclusive, thus coinciding with the special course of lectures by Professor Barr.

It is hoped that some members will avail themselves of this fact and attend both courses.

The fee for the refresher course is £3 3s. It is hoped that arrangements may be made with the committees of management of the Melbourne and Alfred Hospitals, whereby a limited amount of accommodation will be

available at these hospitals for those attending the course at an additional fee of £3 3s. per week to cover board and lodging.

OBSTETRICS COURSE.

The annual obstetrics post-graduate course will be held at the Women's Hospital, Melbourne, from September 24 to October 6, 1934. The course will comprise attendance in all departments of the hospital, with special lectures and demonstrations by members of the staff.

Arrangements have been made whereby a limited number of graduates will be able to enter into residence at the hospital. The fee for the course is £3 3s. The additional fee for accommodation at the hospital will be £3 3s. per week.

Correspondence.

OCULISTS AND OPTOMETRISTS.

Sir: The report of the Ophthalmological Section of the Hobart Congress makes very pitiable reading, if a mere optometrist may be permitted to say so. Surely, at such a meeting, representative of the leading oculists of Australia, one would expect to read of the presentation of papers which opened up new avenues of treatment and discussions on the merits of these proposals.

Instead, we have references to optometrists who dare to criticize the work of oculists, references to "half-baked specialists", and proposals to form a combine of oculists and spectacle makers, the said "combine" to receive the support of the medical profession generally in the recommending of their patients to entrust their eyes to the care of the combine.

Ought it not to have been obvious to these gentlemen that the vitally important thing, the thing which was aimed at their professional probity, was that very criticism of which they complained, any such criticism being essentially a repudiation of their ability to do the work in which they profess to excel? Do they not know that they are not competent in this, the sight-testing department of their practice? That their methods and practices are unreliable, unlikely to lead to the same conclusions if the same eye were tested on different occasions? Could any criticism be more damning than the well known fact that no two of them ever agree in their prescriptions?

"Criticism" is too mild a term to apply to such work as that with which we are familiar and of which the following three prescriptions, issued by three different oculists, afford a fair sample:

1. Right + 2·25 + 1 axis 90. Left + 2·5 + 1 axis 90.
2. Right − 0·75. Left − 0·75.
3. Right − 1·25 − 1·5 axis 90. Left − 1 − 1·5 axis 90.

These should have been:

1. Right + 3·5 + 0·5 axis 115. Left + 3 + 1·25 axis 85.
2. Right − 0·75 cylinder axis 180. Left − 0·75 axis 180.
3. Right − 1·5 − 1·75 axis 115. Left − 1·5 − 1·5 axis 35.

An instance of unreconcilable prescriptions by oculists may also be illuminating; the case was also tested by an optometrist, whose correction is also given.

Oculist A: Right plano. Left − 3·5.
Oculist B: Right + 0·75. Left + 0·5.
Optometrist: Right + 0·5 axis 155. Left + 0·25 axis 155.

Probably every optometrist in Australia could produce batches of similar evidence of the unsoundness of medical sight-testing. In fact, Dr. Cohen's reference to "half-baked specialists" was perhaps a little nearer to the truth than was his intention.

In view of the foregoing, is it not obvious that the proposed "combine" would be an immoral conspiracy, designed to mislead the public into the hands of a group of incompetent practitioners, who would refer them to spectacle makers who were paid "not to criticize"?

In my own Association, at general meetings demonstrations of sight-testing methods are given, members testing each other's eyes and inviting criticism of their methods and results. May I suggest that similar demonstrations be arranged by and for the oculists at the next Congress. In the event of there being a shortage of demonstrators, there should be no difficulty in obtaining the services of numbers of trained optometrists.

Yours, etc.,

A. Knapp.

29, Barrack Street,
Perth,
March 22, 1934.

CHRONIC NEPHRITIS AND LEAD POISONING.

Sir: I and, one fondly hopes, numerous medical men and laymen have read, absorbed and thought carefully over Dr. Jarvis Nye's recently published thesis, "Chronic Nephritis and Lead Poisoning".

One, however, cannot but contemplate the apparent extreme apathy on the part of responsible bodies. Here we have a menace certain and deadly in its action, distributed almost in direct proportion to human habitations.

Only a few weeks ago I saw a boy, aged three and a half years, totally and irrevocably blinded and still manifesting signs of lead poisoning. This child was seen and examined by responsible medical men from north-west Queensland to Sydney. The home in which he lived was painted three years ago with a mixture of white lead and linseed oil, which had become dry and powdery after a year or so from its application.

Cannot something be done to stimulate public action in this matter and prevent further invalidity and premature death in children and adolescents?

One is forcibly struck with the impotent public health activities of local authorities whereby they permit a so-called "health inspector" to "disinfect" houses with a "Flytox" spray after a diphtheria or typhoid patient has been removed.

Would it not be feasible to force lethargic governments into action, obtain legislation and give the "local authorities" some really useful service to perform?

Yours, etc.,

R. H. von der Borch,
Chief Medical Officer,
Mount Isa Mines, Limited.

March 26, 1934.

TUBERCULIN.

Sir: "The unwarranted prejudice which exists against tuberculin should be removed, and the suffering public should be given the benefit of a discovery made fifty years ago" is the opinion expressed by your correspondent, Dr. Fry, March 23, 1934.

Pottenger, nearly a quarter of a century ago, wrote:

> Experience has now so accumulated that tuberculin does do good that the practitioner should look to the best method of making use of it, and not as to whether tuberculin is of use or not; in other words, he should blame his method of use, and not the tuberculin, in case of failure.

Professor Sahli, the Swiss clinician of text book fame, in his treatise on tuberculin treatment, stated some thirty years ago that: "Tuberculin treatment is one of the greatest advances in modern therapeutics."

In the face of such statements, and when we consider the undoubtedly antagonistic attitude of the profession in general towards tuberculin, one cannot help wondering as to wherein lies the reason of it, and especially the reason for the conflicting statements one reads whenever those, evidently periodically occurring, discussions appear in the correspondence columns of medical journals.

Surely there is some misapprehension somewhere if a therapeutic agent which has been before the medical world for half a century is condemned by many and praised by a comparatively few.

To look for a possible solution of the problem let us go back to the beginning of the century; let us again quote Professor Sahli's treatise.

> That tuberculin treatment has been discredited is owing to that mental confusion (which is so evident in the faulty methods of practice) of seeing a resemblance between tuberculin treatment and the serum treatment of diphtheria, tetanus and other diseases.
>
> In order to use tuberculin correctly it must be clearly understood that tuberculin possesses no direct healing power and that it is not an antidote.
>
> Tuberculin is the toxin and not the antitoxin of tuberculosis.
>
> The action of tuberculin depends upon an actively immunizing curative process. A natural healing process is thus initiated by the assimilation of gradually rising doses of tuberculin, which consists of a gradual insensibilization of the body to the chemical tubercle-toxin. This is a tox-immunity.
>
> By this means the "tolerance" of tuberculin in the tuberculous can be raised a million times by a gradual increase of the dose.
>
> This immunizing process is analogous to the natural process of hardening in which, as in connection with many other substances, it is a question of raising the natural capacity in the body of reacting to the tubercle-toxin. These reactions are of an antitoxic nature, but they are of a totally different kind from those on which the serum treatment of diphtheria and tetanus are based.
>
> By means of such increasing reactions the body succeeds not only in tolerating increased quantities of the tuberculin injected, but also in rendering innocuous the toxins produced in the tuberculous foci.
>
> In a word, the stimulation of the local processes of physiological defence, in the tuberculous foci, is the essence of tuberculin treatment.
>
> The aim of tuberculin treatment is, then, the stimulation of that natural healing process which plays the decisive part in the spontaneous cure of tuberculosis, which, we know, is so frequent.
>
> Sahli uses the word "mithridatism" as the general designation of any form of tox-immunity by the progressive assimilation of a poison, as we have it in tuberculin treatment. The expression has its derivation in the Pontic King Mithridates, who, in his fear of being poisoned, rendered himself insensitive by consuming increasing doses of them. When, in his conflict with Pompey, he sought to poison himself and his family, he himself failed, and at his own wish was stabbed by his bodyguard.
>
> In practice the aim of tuberculin treatment is to render a patient tolerant to any such amount of tubercle-toxin (tuberculin) which his own tuberculous foci are pouring into him.

Let us compare this method with that "mental confusion" which has given rise to the use of tuberculin in treatment as if it depended on similar antitoxic actions as those on which the serum treatment of diphtheria and tetanus are based.

Whereas in diphtheria and tetanus a real immunization takes place, the antitoxin neutralising the toxin, in tuberculosis there is no such thing as real immunization.

All attempts to secure immunity in man or beast against living tubercle bacilli by means of tuberculin have proved a failure.

In tuberculosis there is no toxin death, as we find it in pneumonia, typhoid and diphtheria, which diseases, if they run the typical course, are self-limiting by lysis or crisis, on account of the production by the disease of immune bodies.

In tuberculosis death is due to the complete putting out of action of some organ or other in which the tuberculous focus is situated. In the case of tuberculosis of the kidneys a patient will live on until an infinitesimal portion of kidney substance is left, insufficient to meet physiological requirements.

According to Sahli, we can thus summarize. There are two schools of tuberculin treatment: the one which bases its methods of practice on the assumption that tuberculin is of the nature of an antitoxin; the other, which recognizes the absence of immunity in tuberculosis and aims at establishing a state of tolerance by the administration of gradually increasing doses of tuberculin (mithridatism).

The unwarranted prejudice which your correspondent complains of is evidently due to the fact that the former school is still the dominating one, whereas the tolerance school has never been put to the test, and that on account of the unwarranted prejudice which refuses to see any good in tuberculin treatment in any shape or form.

Yours, etc.,

ALFRED E. FINCKH.

The Sydney Clinical Research Laboratories,
227, Macquarie Street,
Sydney,
April 3, 1934.

THE ROYAL AUSTRALASIAN COLLEGE OF SURGEONS.

SIR: I have read with interest the letter of "G.P." in your issue of March 24. The President of the Royal Australasian College of Surgeons is reported to have said: "It was not the policy of the College to object to a general practitioner performing an operation himself, provided he was quite clear as to the possible result." It does not seem to have occurred to the members of the College that they, as a body upon which general practitioners are not represented, are entirely out of order in laying down rules of conduct for general practitioners.

Even in drawing up rules for their own members, where those rules deal with their relations to other practitioners, it would have been correct, as well as courteous, to have consulted the British Medical Association, which represents the great majority of medical practitioners.

I know of at least one rule which appears to carefully conserve the interests of the member, but is grossly negligent in protecting the interests of the patient or the general practitioner referring the case.

If the College of Surgeons persists in telling the general practitioner what his conduct should be, the time is not very far distant when the general practitioner will ask by what authority the College of Surgeons so presumes, and may offer a few suggestions as to the conduct of members of the College in their relations to general practitioners.

If a statement similar to that partially quoted above had come officially from the President of the Federal Council of the British Medical Association, it would have been an entirely different matter.

Yours, etc.,

GIFFORD CROLL.

Brisbane,
April 6, 1934.

VITAMIN C.

SIR: In reply to F. J. Bridges's comment (in the issue of March 31, 1934) on my paper on vitamin *C* (in the issue of March 10, 1934), I venture to correct his statement concerning William Harvey's cure of scurvy in rats. As far as I am aware, scurvy cannot be induced in rats, and it is assumed, in lieu of other hypothesis, that the animals are capable of synthesizing the vitamin.

Breidahl and myself have not succeeded in inducing scurvy in rats, or even in reducing the vitamin *C* reaction of the adrenal in rats which have been for two to three weeks on a scorbutic diet, which had given guinea-pigs definite symptoms of scurvy in the same time and had caused their adrenals to lose entirely their vitamin *C*.

Yours, etc.,

GEOFFREY BOURNE.

The University of Melbourne,
Melbourne,
April 13, 1934.

Obituary.

ST. JOHN WARBURTON DANSEY.

We regret to announce the death of Dr. St. John Warburton Dansey, which occurred on April 22, 1934, at Rose Bay, New South Wales.

Books Received.

THE 1933 YEAR BOOK OF UROLOGY; 1934. Chicago: The Year Book Publishers. Crown 8vo., pp. 445, with illustrations.

THE STUDENTS' POCKET PRESCRIBER AND GUIDE TO PRESCRIPTION WRITING, by D. M. Macdonald; Tenth Edition; 1934. Edinburgh: E. and S. Livingstone. Demy 18mo., pp. 263. Price: 3s. net.

HANDBOOK OF THERAPEUTICS, by D. Campbell, M.A., B.Sc., M.D., F.R.F.P.S.; Second Edition; 1934. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 464, with illustrations. Price: 12s. 6d. net.

Diary for the Month.

May 1.—Tasmanian Branch, B.M.A.: Council.
May 2.—Western Australian Branch, B.M.A.: Council.
May 2.—Victorian Branch, B.M.A.: Branch.
May 3.—South Australian Branch, B.M.A.: Council.
May 4.—Queensland Branch, B.M.A.: Branch.
May 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
May 8.—Tasmanian Branch, B.M.A.: Branch.
May 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
May 11.—Queensland Branch, B.M.A.: Council.
May 15.—New South Wales Branch, B.M.A.: Ethics Committee.
May 15.—Tasmanian Branch, B.M.A.: Council.
May 16.—Western Australian Branch, B.M.A.: Branch.
May 22.—New South Wales Branch, B.M.A.: Medical Politics Committee.
May 23.—Victorian Branch, B.M.A.: Council.
May 24.—New South Wales Branch, B.M.A.: Clinical Meeting.
May 25.—Queensland Branch, B.M.A.: Council.
May 31.—South Australian Branch, B.M.A.: Branch.
May 31.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. G. Forsyth (B.M.A.) has been appointed Certifying Medical Practitioner at Horsham, Victoria, pursuant to the provisions of the *Workers' Compensation Act*, 1928.

* * *

Dr. A. Adams has been appointed Medical Officer of Health to the Onslow Road Board, Western Australia.

* * *

Dr. J. Grahame Drew (B.M.A.) has been appointed Deputy Commissioner of Public Health and Deputy Inspector, School of Anatomy, Queensland, in pursuance of the provisions of *The Public Service Acts*, 1922 *to* 1924, and *The Health Acts*, 1900 *to* 1931.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv and xv.

Alfred Hospital, Melbourne, Victoria: Officer-in-Charge of General Clinic.

Children's Hospital, Carlton, Victoria: Assistant Surgeons.

The Brisbane and South Coast Hospitals Board, Queensland: Masseuse.

The Public Service Board, Sydney, New South Wales: Assistant Medical Officer (female).

The University of Melbourne, Victoria: Demonstrator in Clinical Physiology.

Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
New South Wales: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Orford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
Victorian: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
South Australian: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
Western Australian: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
New Zealand (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

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